CANADIAN THESES ON MICROFICHE

I.S.B.N.

THESES CANADIENNES SUR MICROFICHE

National Library of Canada
Collections Development Branch

Canadian Theses on
Microfiche Service

Ottawa, Canada
K1A 0N4

Bibliothèque nationale du Canada
Direction du développement des collections

Service des thèses canadiennes
sur microfiche

NOTICE

The quality of this microfiche is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us a poor photocopy.

Previously copyrighted materials (journal articles, published tests, etc.) are not filmed.

Reproduction in full or in part of this film is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30. Please read the authorization forms which accompany this thesis.

THIS DISSERTATION
HAS BEEN MICROFILMED
EXACTLY AS RECEIVED

AVIS

La qualité de cette microfiche dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de mauvaise qualité.

Les documents qui font déjà l'objet d'un droit d'auteur (articles de revue, examens publiés, etc.) ne sont pas microfilmés.

La reproduction, même partielle, de ce microfilm est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30. Veuillez prendre connaissance des formules d'autorisation qui accompagnent cette thèse.

LA THÈSE A ÉTÉ
MICROFILMÉE TELLE QUE
NOUS L'AVONS REÇUE

NL-339 (r. 82/08)

Canada

0-315-05990-7

National Library of Canada | Bibliothèque nationale du Canada

Canadian Theses Division | Division des thèses canadiennes

Ottawa, Canada
K1A 0N4

53911

PERMISSION TO MICROFILM — AUTORISATION DE MICROFILMER

• Please print or type — Écrire en lettres moulées ou dactylographier

Full Name of Author — Nom complet de l'auteur

Marnie Jean Finstad

Date of Birth — Date de naissance	Country of Birth — Lieu de naissance
Feb. 4, 1950	Canada

Permanent Address — Résidence fixe

11122-171 Ave.
T5G 0A4
Edmonton, Alberta

Title of Thesis — Titre de la thèse

Dynamic Factors Involved in Biofeedback and Relaxation Therapy of Migraine Headache

University — Université

University of Alberta

Degree for which thesis was presented — Grade pour lequel cette thèse fut présentée

M.Ed.

Year this degree conferred — Année d'obtention de ce grade	Name of Supervisor — Nom du directeur de thèse
1981	Dr. G.C. Fitzsimmons

Permission is hereby granted to the NATIONAL LIBRARY OF CANADA to microfilm this thesis and to lend or sell copies of the film.

The author reserves other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

L'autorisation est, par la présente, accordée à la BIBLIOTHÈQUE NATIONALE DU CANADA de microfilmer cette thèse et de prêter ou de vendre des exemplaires du film.

L'auteur se réserve les autres droits de publication; ni la thèse ni de longs extraits de celle-ci ne doivent être imprimés ou autrement reproduits sans l'autorisation écrite de l'auteur.

Date	Signature
Oct. 10, 1981	Marnie D. Finstad

NL-91 (4/77)

THE UNIVERSITY OF ALBERTA

DYNAMIC FACTORS INVOLVED IN BIOFEEDBACK AND RELAXATION THERAPY OF MIGRAINE HEADACHE

by

© Marnie Jean Finstad

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF MASTER OF EDUCATION

DEPARTMENT OF EDUCATIONAL PSYCHOLOGY

EDMONTON, ALBERTA

FALL, 1981

THE UNIVERSITY OF ALBERTA

RELEASE FORM

NAME OF AUTHOR Marnie Jean Finstad

TITLE OF THESIS Dynamic Factors Involved in Biofeedback and Relaxation Therapy of Migraine Headache

DEGREE FOR WHICH THESIS WAS PRESENTED Master of Education

YEAR THIS DEGREE GRANTED 1981

Permission is hereby granted to THE UNIVERSITY OF ALBERTA LIBRARY to reproduce single copies of this thesis and to lend or sell such copies for private, scholarly or scientific research purposes only.

The author reserves other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.

(Signed) Marnie Finstad

PERMANENT ADDRESS

11122 - 71 Avenue
Edmonton, Alberta
T6G 0A4

DATED Oct. 15, 81

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled Dynamic Factors Involved in Biofeedback and Relaxation Therapy of Migraine Headache submitted by Marnie J. Finstad in partial fulfilment of the requirements for the degree of Master of Education in Educational Psychology.

Supervisor

DATE 1 October 1981

ABSTRACT

Migraine headache is a functional disorder of vascular mechanism which is known to affect a sizeable proportion of the population in our Western civilization. Migraine has been treated, traditionally, by pharmacotherapy, and more recently, through behavioural stress management programs. This study applies behavioural techniques through the use of biofeedback instrumentation and relaxation training. The subjects were forty-eight volunteers, all of whom suffered from migraine type headaches. Biofeedback was applied in two modalities, through electromyographic feedback from the frontalis muscle, or by digital skin temperature feedback from the palmar surface of the third finger.

A two (treatments) by two (groups) design across repeated measures was used. Repeated measures included pre-treatment, treatment, and post-treatment periods. Subjects in both treatment modalities were trained to a preset experimental criterion. Following attainment of criterion, subjects were trained to apply their skill without visual or auditory feedback. Headache activity was recorded daily through the experimental period.

The procedures were found to be effective overall in bringing about a reduction in headache activity. Those subjects who were trained by muscular relaxation procedures experienced a greater headache reduction than those who were trained to control peripheral skin temperature. Subjects who were successful in attaining the preset experimental criterion did not experience a greater reduction in headache activity than those who did not. Psychological factors are considered to influence success in biofeedback training and subsequent headache reduction. The experimental results suggest implications for future treatment and research.

ACKNOWLEDGEMENTS

I would like to thank my supervisor, Dr. G. W. Fitzsimmons, for his continued encouragement and support throughout the past year. I would also like to thank the other members of my committee, Dr. H. W. Zingle and Dr. D. P. Hube, for their assistance and participation.

I would like to express thanks, appreciation and gratitude to Penny Ford, a valued friend and fellow researcher, and also thank Scott Sellick for his participation in the study. I wish to thank Mrs. Faye Carbol and Dr. Pat Carney for their willingness to share their experience and knowledge.

Acknowledgement is given to Dwight Harley for his assistance in the statistical analysis, and to Udaya Dash for his help in statistical interpretation. Recognition and appreciation is also given to Mrs. Sheri McLean for her patience and her typing skill.

A special acknowledgement is given to the 'migraineurs', who by sharing their stresses, their pain, and their efforts towards relief, made this study possible.

TABLE OF CONTENTS

CHAPTER		PAGE
1.	INTRODUCTION	2
	1.1 Overview	2
	1.1.1 Purpose of Study	2
	1.1.2 Chapter Outline	2
	1.1.3 General Rationale	4
2.	THE MIGRAINE HEADACHE	7
	2.1 Definition	7
	2.1.1 Classification	8
	2.1.2 Symptomology	9
	2.1.3 Epidemiology	11
	2.2 Physiology	12
	2.2.1 Vascular Mechanisms	12
	2.2.2 Biochemical Factors	16
	2.3 Etiology	20
	2.3.1 External Precipitators	20
	2.3.1.1 Foods	20
	2.3.1.2 Hormonal Triggers	21
	2.3.1.3 Other Factors	22
	2.3.2 Internal Susceptibility	22
	2.3.2.1 Genetic Predisposition	22
	2.3.2.2 Personality Correlates	23
	2.3.3 The Stress Reaction	26
	2.4 Treatment Modalities	30
	2.4.1 Identification and Avoidance of Triggers	30
	2.4.2 Drug Therapy	30
	2.4.3 Psychotherapy	32
	2.4.4 Relaxation Training	34
	2.5 Summary	35
3.	PROPOSED MECHANISMS OF BIOFEEDBACK TREATMENT	38
	3.1 Overview	38
	3.2 Biofeedback as a Behavioral Therapy	38

CHAPTER PAGE

3.3 The Process of Biofeedback 39

3.3.1 Biofeedback as Applied to Temperature Training 40

3.3.1.1 The Autonomic Nervous System 40
3.3.1.2 Voluntary Control of Skin Temperature 41
3.3.1.3 Experimental Applications 45
3.3.1.4 Summary 47

3.3.2 Biofeedback as Applied to Electromyography 48

3.3.2.1 The Occipito-frontalis Muscle 48
3.3.2.2 Voluntary Control of Muscle Activity 49
3.3.2.3 Experimental Applications 50
3.3.2.4 Summary 52

3.4 Non-Specific Learning Components (Placebo Effects) 53

3.5 Summary 56

3.6 Experimental Rationale 57

3.7 Research Questions 58

4. METHOD 60

4.1 Subjects 60

4.2 Apparatus & Facilities 61

4.3 Research Design 65

4.4 Procedures 66

4.4.1 Stress Profile Procedure 66

4.4.1.1 Differential Recovery Calculations 67

4.4.2 Treatment Procedure 68

4.4.2.1 Group 1: EMG Training 68
4.4.2.2 Group 11: Skin Temperature Training 70

4.4.3 Follow-up Procedure 71

CHAPTER		PAGE
5.	RESULTS	73
	5.1 Changes in Headache Intensity: Effects of Training Method and Group-reactivity	73
	5.1.1 Reactivity and Treatment Groupings	73
	5.1.2 Headache Intensity Variable	74
	5.1.3 Analysis of Variance	77
	5.1.4 Summary	81
	5.2 Reduction in Headache Intensity from Pre- to Post-treatment in Relation to Training Method and Criterion-attainment	83
	5.2.1 Criterion Measures - EMG and Temperature	83
	5.2.2 Statistical and Descriptive Analyses	83
	5.2.3 Summary	90
6.	DISCUSSION	92
	6.1 Overview	92
	6.2 The Research Questions	92
	6.2.1 Question One	92
	6.2.1.1 Summary	96
	6.2.2 Question Two	97
	6.2.2.1 Summary	99
	6.2.3 Question Three	99
	6.2.3.1 Summary	104
	6.3 Practical and Theoretical Implications	104
	6.4 Related Research	106
References		107
Appendices		114

LIST OF TABLES

TABLE		PAGE
4-1	Characteristics of Study Population	62
5-1	Distribution of Subjects by Groups and Treatment Methods	76
5-2	Mean Headache Levels Across Reactivity Groups, Treatments and Periods	78
5-3	Summary of Analysis of Variance for Reactivity Grouping, Treatment Variable, and Headache Intensity Variable, Across Three Factor Repeated Measures	79
5-4	Treatment and Period Means as Measured with Group Effect Collapsed and Group and Period Means as Measured with Treatment Effect Collapsed	82
5-5	Summary of Analysis of Variance for Treatment Variable, Attainment (non-attainment) of Criterion, Headache Intensity Variable, Two Factor ANOVA	84
5-6	Pearson Product-moment Correlation Coefficients Based on a Comparison of the Difference Between Pre- and Post-Baseline EMG (E1-E3) Measurements and Pre- and Post-Baseline Temperature (T2-T1) Measurements and Headache Activity Reduction (P1-P3)	85
5-7	Mean EMG and Temperature Baseline Levels as Measured during Final Five Minutes of Relaxation Prior to Stressing in Pre (P1) and Post (P3) Treatment Periods	89

LIST OF FIGURES

FIGURE		PAGE
2-1	Hypothesized Biochemical and Vascular Mechanisms of a Migraine Attack	19
5-1	A Scattergram of EMG and Temperature Reactivity T-scores	75
5-2	Mean Headache Activity Levels for the Two Treatments (EMG, temperature). Two Groups (EMG reactive and temperature reactive) and Three Periods (P1=pre-treatment, P2=treatment, P3=post-treatment)	80
5-3	Mean Headache Activity Reductions (P1-P3) for the Two Treatments (EMG, temperature) for Subjects who Attained Criterion and for Subjects who did not Attain Criterion	87

CHAPTER ONE

I. INTRODUCTION

1.1 Overview

1.1.1 Purpose of Study

1.1.2 Chapter Outline

1.1.3 General Rationale

CHAPTER ONE

1. INTRODUCTION

1.1 Overview

1.1.1 Purpose of Study

The primary purpose of this study was to investigate the effects of a behavioral training program in the alleviation of migraine headache. The behavioral modification is effected through two procedures, biofeedback training and relaxation training. Biofeedback training was effected in two modalities. One half of the subjects investigated received electromyographic biofeedback training and progressive muscle relaxation instructions. The remaining subjects received digital skin temperature biofeedback training and were taught to relax using autogenic techniques. All biofeedback training was directed to a preset criterion. If this criterion was realized, weaning procedures were initiated to train the subject to maintain his/her newly learned relaxation techniques without external feedback. Follow-up measures were included in the study to monitor the continuing effects of the program on headache intensity and frequency.

1.1.2 Chapter Outline

Chapter One serves as an introduction to the study. Included is a chapter outline and a brief introduction to the general rationale underlying the use of biofeedback and relaxation training in migraine headache therapy.

Chapter Two, 'The Migraine Headache', examines the migraine syndrome. This examination begins with a definition of migraine in terms of symptomology and medical classification schemes. The physiology of the migraine

is discussed next, in mechanical and biochemical aspects. Etiology is examined in terms of specific initiating factors, external and internal. These specific triggering mechanisms are then placed within the framework of the general stress response. The chapter closes with a delineation of treatment modalities, which include environmental control, drug therapy, behavioral counselling to modify psychological propensity, and relaxation training.

Chapter Three, 'Biofeedback and Migraine Headache', reviews the basic functioning of the autonomic nervous system and of the action of striated muscle, both of which may be involved in the migraine syndrome. Voluntary control of the autonomic nervous system is related to skin temperature biofeedback training, and experimental applications of such training are examined. This discussion is followed with a description of biofeedback as applied in the voluntary control of muscular activity, with an examination of related studies. A discussion of non-specific learning components in the training process is included. These uncontrolled variables are seen as psychological influences which act as modifying factors to the measured physiological changes. A summary of this chapter leads into a specific treatment rationale and to the three specific questions to be considered in the study.

Chapter Four, 'Method' reviews the experimental procedures employed in the study.

Chapter Five, 'Results', presents the experimental design, the data, the method of analysis, and the results of the analyses as applied to the two questions under study which require statistical interpretation. Observations as related to question three are anecdotal, and are reserved until Chapter Six.

Chapter Six, 'Discussion' begins with a brief overview followed by a discussion related directly to each of the research questions. The Chapter concludes with a discussion of results in relation to some of the theoretical and practical implications of biofeedback training in migraine headache, and a discussion of implications for further research.

1.1.3 Biofeedback and Migraine Headache: General Rationale

Biofeedback is a technique which involves the use of electronic equipment to provide to a human subject feedback of some internal physiological event. The feedback is commonly provided in the form of a visual or auditory signal (Basmajian, 1979).

Teaching human subjects to control physiological processes has had therapeutic results in a number of areas, one of which includes the alleviation of migraine through teaching patients to raise finger-tip temperature. Temperature training has frequently been associated with autogenic relaxation, a procedure developed by Schultz and Luthe in Germany (1969), whereby a subject repeats relaxing, self-warming statements to himself. Chronic muscle tension headache has been treated successfully through myoelectric feedback which is aimed at reducing general muscle tension. Jacobson (1933) first developed the technique in the 1920's. He developed a progressive somatic relaxation procedure which he monitored with primitive electromyographic equipment.

An overactive sympathetic nervous system seems to be implicated in the pathogenesis of migraine. Migraines are frequently accompanied by tension headaches which are characterized by excessive activity in the muscles of the scalp, forehead and neck.

Evidence seems to suggest that training with EMG feedback for muscular relaxation and/or skin temperature feedback to augment peripheral

vasodilation and concommitant relaxation is effective in reducing headache intensity and frequency. The present study is conducted to examine the relative efficacy of the two biofeedback methods, EMG and digital temperature training, as applied to subjects who suffer from headaches of the migraine type. To date, neither method has been demonstrated to be unequivocally superior to the other in alleviating headache symptoms, nor has the most efficacious treatment procedure been demonstrated. Experimental evidence, which may indicate one method to be superior to the other, is seen to be of value in future treatment of migraine headache.

CHAPTER TWO

2. THE MIGRAINE HEADACHE

2.1 Definition

2.1.1 Classification
2.1.2 Symptomology
2.1.3 Epidemiology

2.2 Physiology

2.2.1 Vascular Mechanisms
2.2.2 Biochemical Factors

2.3 Etiology

2.3.1 External Precipitators

2.3.1.1 Foods
2.3.1.2 Hormonal Triggers
2.3.1.3 Other Factors

2.3.2 Internal Susceptibility

2.3.2.1 Genetic Predisposition
2.3.2.2 Personality Correlates

2.3.3 The Stress Reaction

2.4 Treatment Modalities

2.4.1 Identification and Avoidance of Triggers
2.4.2 Drug Therapy
2.4.3 Psychotherapy
2.4.4 Relaxation Training

2.5 Summary

CHAPTER TWO

2. THE MIGRAINE HEADACHE

2.1 Definition

Headache is a unique syndrome in medicine and is considered to be the most common medical complaint of civilized man, and yet, severe headache is only rarely caused by organic disease. In a small percentage of cases, headache may be the presenting complaint of severe disease such as brain tumor, cerebral hemorrhage or meningitis, and in consideration of this possibility every headache patient should have a careful physical and neurological examination (Diamond & Dalessio, 1978). But in the majority of cases, where no organic lesion is found, headache is seen to be a physical symptom of inner distress, a somatic expression of unresolved conflict, repressed tension, a disharmony of mind and body, and yet, the pain of headache may be equally intense, whether its primary source is organic or psychological.

Physicians have separated headaches into three categories; vascular, muscle contraction, or traction and inflammatory headache. Traction and inflammatory headaches are classified as those headaches which do have an organic basis, such as tumor, a hematoma or an abscess in the skull. A muscle contraction headache is, as its name implies, characterized by persistent and painful contractions of the head, face and neck. These headaches may persist for long periods of time and are considered to be associated with depression or anxiety. Vascular headaches may be of the migrainous or non-migrainous type. Both types involve dilatation of cranial arteries, but the non-migrainous vascular headache is non-recurring and is caused by a specific irritant such as a systemic infection or a noxious chemical (Diamond & Dalessio, 1978).

In summary, migraine is seen to be a common and distressing malady which affects civilized man. The prevalence and the severity of the migraine attack suggest the loss of many work hours and the endurance of many hours of personal pain. Studies of the incidence of migraine have been carried out in Scandinavia, Britain and the United States. No information is available regarding the incidence of migraine in the Asian or African countries. If cultural differences exist, such comparative data would be of much interest in relation to the causal factors underlying migraine.

2.1.1 Classification

Migrainous headaches are recurring headaches of the vascular type. The Ad Hoc Committee on Classification of Headache (1962) groups these headaches into five classes.

A. The 'Classic' migraine which presents a warning phase characterized by visual or sensory phenomena, followed by a pulsating unilateral head pain. This type includes approximately 10% of migraine sufferers (Friedman, 1975).

B. The 'Common' migraine is a vascular headache, which may be bilateral and which does not present the striking sensory prodomes, although less sharply defined warning signs may be present. This headache has been referred to as 'atypical migraine' or 'sick headache' and is the most common form of migraine.

C. 'Cluster' headache is a vascular headache, which is usually unilateral, brief in duration but which will occur in clusters or groups over a period of several weeks.

D. 'Hemiplegic' migraine and 'opthalmoplegic' migraine are rare forms of migraine which are distinguished by sensory sensations,

muscular weaknesses or paralysis which are maintained for a period during and after the headache.

E. 'Lower half' headache is a headache which is felt primarily in the lower half of the face, and which includes some facial neuralgias, a neuralgia being a pain which will extent along the course of a nerve.

This classification scheme is based on the pain mechanisms of migraine and provides a logical framework which is useful for diagnosis to the medical profession, and for those who study migraine.

2.1.2 Symptomology

Although the 'headache' is only a part of a widespread disturbance, it is the most distinctive and readily apparent aspect. Migraine headache was described and named 2000 years ago by Arataeus of Cappodocia (Sachs, 1971). The word 'migraine' comes from the French through the Greek word 'hemikrania', meaning pain or aching on one half of the head. Migraine is distinguished by this feature of severe, throbbing unilateral head pain, frequently associated with nausea, and periodic in nature.

Wolff (1963) has described the symptomology of migraine with a thoroughness that has yet to be surpassed. Included in this description, Wolff described the variability of the pre-headache phenomena. He reports that many patients experience a strong sense of well being and high spirits before the onset of an attack, which may be exhibited by talkativeness and increased appetite. For the small group who experience what is classified as classical migraine, this period is followed by visual and other sensory disturbances immediately before the onset of the headache. The visual disturbances may occur as blind spots, bright flashes of light, or geometric designs which pass

across the visual field. These visual effects are not always followed by headache. They may last from a few minutes upwards to an hour, and usually terminate before the headache begins. There may be a concurrent numbness, or prickling sensations in the face and unilateral hand, speech affects or hallucinations of movement, nausea, sweating, or fainting. Patients who experience 'common migraine' have less clearly defined warning syndromes. They may experience feelings of tension, hunger, wakefulness, depression or simply loss of energy. The patient is apt to be extremely unsocial, hostile and destructive during this period. In addition, memory, attention and concentration may be poor, and these mood changes may continue through the duration of the attack.

The pain itself, while frequently unilateral, may also be bilateral. Initially, the quality of the headache is a throbbing and aching which may become a steady ache after some hours. The intensity of the pain varies for each individual and between individuals, from very slight to an intolerable, fierce pain which causes prostration and near coma. This pain is intensified by movement, bodily effort, or position change. The intensity of the pain is also affected by bright light, loud sounds or by mental effort (Wolff, 1963).

The duration of the attack may be anywhere from a few minutes to several days. Attacks are often observed to last from 'sun-up' to 'sun-down', but Wolff noted that migraineurs will also frequently wake up with a headache. Attacks may occur several times a week, once a month, or very infrequently. Migraine headache attacks will occasionally occur during a 'let down' period such as a week-end, the first days of a holiday or at the time of a planned social engagement.

The site of the headache is most commonly in the frontal and temporal or lateral regions of the head, but a piercing, sharp pain may settle behind the eye, and the pain can radiate across the head and into the face and neck (Diamond & Dalessio, 1978).

Wolff pointed out, further, that a migraine patient looks and feels ill. The skin may exhibit a pallor and poor tone. Features will look strained and imply suffering. The patient is apt to be tearful, tired and irritable, and will complain of feeling chilly, particularly in the extremities. The temporal, frontal and supraorbital blood vessels on the painful side appear distended and are painful to touch, as is the entire scalp area. Anorexia or lack of appetite and nausea accompany most headaches. Vomiting may occur throughout the attack and may be a serious aspect, causing a delay in recovery. Other symptoms may include nasal constriction, abdominal distention, constipation, fever, disturbances in water metabolism, and photophobia (an undue sensitivity to light). After an attack, usually the patient feels euphoric, filled with energy and drive, enthusiastic and relaxed.

Except on rare occasions all symptoms disappear at the end of the attack. In 1-2% of migraine sufferers, symptoms will continue beyond the headache period, as in hemiplegic or opthalmoplegic migraine. These complications include muscular weakness, visual defects and rarely a hemiparalysis. It is presumed that these changes are related to a very prolonged vasoconstriction in the migraine attack (Wolff, 1963).

2.1.3 Epidemiology

Migraine is commonly reported to occur in approximately 10% of

the population, a frequently quoted figure which does not seem to be based on any particular survey. Some recent surveys report a much higher incidence. Waters and O'Connor (1975) studied large numbers of population groups with standardized questionnaires administered in three separate epidemological surveys, and cited prevalence rates of migraine type headaches of 23-29% in women and 15-20% in men. Ziegler (1978)stated that his own surveys indicated that 20-25% of the whole population suffers from a migraine type of headache. Variations in these figures may be a result of difficulties in classification, poor response rates to surveys, or a neurotic tendency of patients in admitting or not admitting to symptoms, or a combination of these factors (Ziegler, 1978). Women appear to experience migraines more commonly than men. Water's and O'Connor's study (1975) showed that more women than men have headaches and that in both sexes the incidence of headache decreases with age. Henryk-Gutt and Rees (1973) in surveys conducted through the civil service, indicate that women report about twice as many headaches as do men. The sex differences in the occurrence of migraine suggest a relationship to hormonal factors, as does the occurrence of 'menstrual migraine' (Ziegler, 1978). These results are confirmed by Ziegler's report of his own survey (1978). Waters (1971) also reported no relationship between intelligence and social class and migraine, as has been previously suggested. He does suggest that the more intelligent, and persons from upper social classes, are more apt to consult a doctor concerning their headaches.

2.2 Physiology

2.2.1 Vascular Mechanisms

The vascular and pain mechanisms in migraine are well described by

Rose (1979). The structures within the head that are sensitive to pain are the blood vessels and the coverings of the brain (the meninges). Brain substance itself is insensitive to pain, and neurosurgeons can operate on the brain without anaesthesia. The pain felt in a migraine headache is in part due to the pressure of swollen blood vessels against the sensitive membranes that sheath the skull (Rose, 1979).

The brain is enclosed by a container of bone (the skull) which is itself covered by a fibrous structure and skin (the scalp). Beneath the skin are many blood vessels, including arteries which take blood to the heart, and veins which return blood to it. The scalp arteries come out of the large carotid artery which divides in the neck into two branches. The external carotid sends blood to the outside of the skull and to the brain coverings (the meninges) while the other branch, the internal carotid, enters the skull and joins with vessels from the vertebral artery. These arteries provide the blood supply to the brain.

The brain has three coverings or meninges, the dura mater, the outer covering, the arachnoid mater underlying it, and the inner covering, the pia mater. Between the arachnoid and the pia is a fluid, the cerebral spinal fluid, which bathes and protects the brain and spinal cord.

The skull itself has a covering, the periosteum, with a porous inner structure which contains short blood vessels. The periosteum does have pain receptors, and when it is stretched by inflammation, will cause severe pain. All the main arteries to the dura and some smaller branches are pain sensitive, and stretching of these vessels

will cause severe pain. Parts of the dura are also very sensitive to pain and pain produced in the frontal area may spread to behind the eye. The great venous sinuses are very sensitive to pain as are the main arterial blood vessels. Dilatation of the internal carotid inside the skull will cause a dull and throbbing pain that eventually may become nauseating. This pain is localized behind the eye and over the temple on the same side as the affected artery. There are twelve cranial nerves which arise from the brain, but all the pain impulses in the head are carried by only two of these, the fifth and the ninth cranial nerves, which also carry most of the other sensations felt in the head and face.

Most blood vessels, including arteries, have a smooth muscular coat which enables them to change their diameter. These changes alter the amount of, and the resistance to blood flow. The throbbing pain of vascular headaches, including migraine, is in part caused by the stretching of these blood vessels (Rose, 1979). Persistent dilatation causes these vessels to become rigid and pipe-like. Pain at this stage is a steady ache, which replaces the earlier throbbing. During this time, a contraction of the neck muscles usually occurs and a muscle-contraction pain develops. This spasm is a reaction to the intense vascular pain and may endure after the vascular symptoms have subsided (Friedman, 1954).

Preceding the headache phase of migraine, there is a narrowing of blood vessels (vasoconstriction) inside the head. This decrease in blood flow accounts for the visual and other prodromal symptoms before an attack. This theory of prodromal vasoconstriction initially put forth by Wolff (1963) has been subsequently verified by cerebral

arteriography during and following the migraine prodrome (Dukes & Vieth, 1964).

The smooth muscles covering the blood vessels are supplied by nerves which have functions similar to those of nerves going to other muscle fibres, stimulation of which will cause the muscles to contract and narrow the blood vessels. These nerves come from a part of the nervous system that is not usually under the control of the conscious mind, called the autonomic nervous system. This system has two parts, the sympathetic and the para-sympathetic, each of which have different and opposing actions. The sympathetic branch increases the tension or tone of blood vessels, and thereby increases resistance to increases in blood pressure. The sympathetic system alters the blood vessel calibre in such a way, so that there is a decrease in the blood flow to the periphery of the body, and an increase in the blood flow to the trunk and head. This is the system which takes over in situations of danger, in what has come to be known as the 'fight-or-flight response' (Rose, 1979).

In summary, the mechanical aspects of migraine include an initial intracranial vasoconstriction phase. During this period, insufficient blood supply and a consequent reduction in tissue oxygenation may cause sensory aberrations. The period of intracranial vasoconstriction is followed by a period of extracranial dilatation and it is during this period that the pulsing, throbbing pain of migraine is experienced. During this phase, the migraine sufferer may experience cold in the extremities. Such symptoms suggest that the blood pool has been shifted to the brain and torso, and away from the periphery. This vascular process is initiated by the sympathetic branch of the autonomic

nervous system, a system which is responsive to 'stress'. Implied in this description, is the rationale for biofeedback assisted skin temperature training procedures which may act specifically to initiate a hydraulic shift of the blood pool away from the brain and torso and to extremities, thereby reducing the pressure and pain in the strained vessels. Such training may also act to initiate a reversal of the more general stress response.

2.2.2 Biochemistry

The dilatation of blood vessels alone is not sufficient to produce headache. Other dynamic or chemical factors contribute (Friedman, 1954). A number of these specific vasoactive biochemical substances have been identified in association with migraine attacks by a series of investigators. Some of the important findings in these investigations are listed below.

While the tone of the large blood vessels is controlled primarily by neurogenic stimulation, the micro circulation, which includes the capillaries and arterioles, is controlled by circulating substances in the plasma, which influence the tone of vascular smooth muscle and alter the calibre of its vessels (Anthony & Lance, 1975). Anthony and Lance (1967) found levels of platelet serotonin (5-hydroxytriptamine) to be elevated in the pre-headache phase and decreased during the headache period. Platelets are minute circular discs found in the blood of all animals, and are concerned with blood flow and blood coagulation. Platelets contain 98.5% of blood serotonin. Anthony and Lance (1975) further report that a fall in plasma levels of serotonin occurred in 85% of migraine attacks investigated, during the actual attack phases. In 1961, Sucuturi found that urinary 5-hydroxyindoleacetic acid (5-HIAA),

the major catabolic product of serotonin, was increased during a migraine attack. Anthony (1976) found higher levels of free fatty acids to be found in patients when a migraine headache is present. Platelet aggregation has been induced in vitro by free fatty acids, and such aggregation is associated with the release of histamine and serotonin. Therefore, certain free fatty acids may be a causative factor in releasing serotonin from the platelets. Chapman et al. (1960) report the presence of increased levels of vasoactive polypeptides (neurokinins) during a migraine attack. These substances act in the blood vessels and perivascular tissue to reduce pain thresholds, to increase capillary permeability and to induce local inflammation. Plasmakinins are released by neurostimulation or stress of the central nervous system. Sandler (1977) found a significant decrease in blood levels of serotonin (5-HT) and of platelet monamine oxidase during migraine headache attacks, monamine oxidase (MAO) being an enzyme which catalyzes the breakdown of 5-HT. Horrobin (1977) found that increasing levels of substances known as prostaglandins produce vasoconstriction followed by vasodilatation. The release of prostaglandins from the lungs is stimulated by serotonin and by tyramine, and prostaglandins are in turn involved in platelet aggregation and serotonin release.

Anthony and Lance (1975) consolidate this information and present a hypothesis regarding the biochemical and vascular mechanisms of a migraine attack. An unknown 5-HT releasing factor appears in the blood at the onset of the attack (possibly free fatty acids). Increased levels of 5-HT in the blood then produce vasoconstriction (causing prodromal symptoms). As this released 5-HT is excreted or

metabolized, blood levels fall rapidly, and its influence on blood vessel tonus is lost. Vasodilation of the scalp arteries and constriction of skin capillaries then occurs. It is theorized that these rapid 5-HT fluctuations may stimulate the vomiting center in the medulla. The 5-HT releasing factor may also prevent the platelets from picking up more 5-HT from the gut, as platelet aggregability remains high throughout the attack and appears to be inversely proportional to serotonin levels in the blood. The pain and edema of the arterial walls may be partly due to the local release of prostaglandins. Fanchamps (1974) introduced the involvement of plasmakinins (or neurokinins as named by Wolff), which along with histamine and 5-HT are known to provoke local pain and lower the pain threshold. Fanchamps included histamine as a precipitating factor. Histamine is released by mast cells in the vessel walls along with 5-HT during the migraine attack. Mast cells are connective tissue cells whose specific physiologic function is unknown. Histamine produces further vasodilation and increases capillary permeability. This process is shown schematically in Figure 2-1, a schema partially derived from the work of Anthony and Lance (1975).

In summary, it is concluded that biochemical substances are somehow released in excess by certain stressors in susceptible persons, and that these substances act in conjunction with one another to produce painful vascular dilation, inflammation, a lowering of local pain thresholds, and hence, a headache. The release of serotonin (5-HT), and its subsequent depletion appears to be a primary factor in the constriction-dilatation sequence.

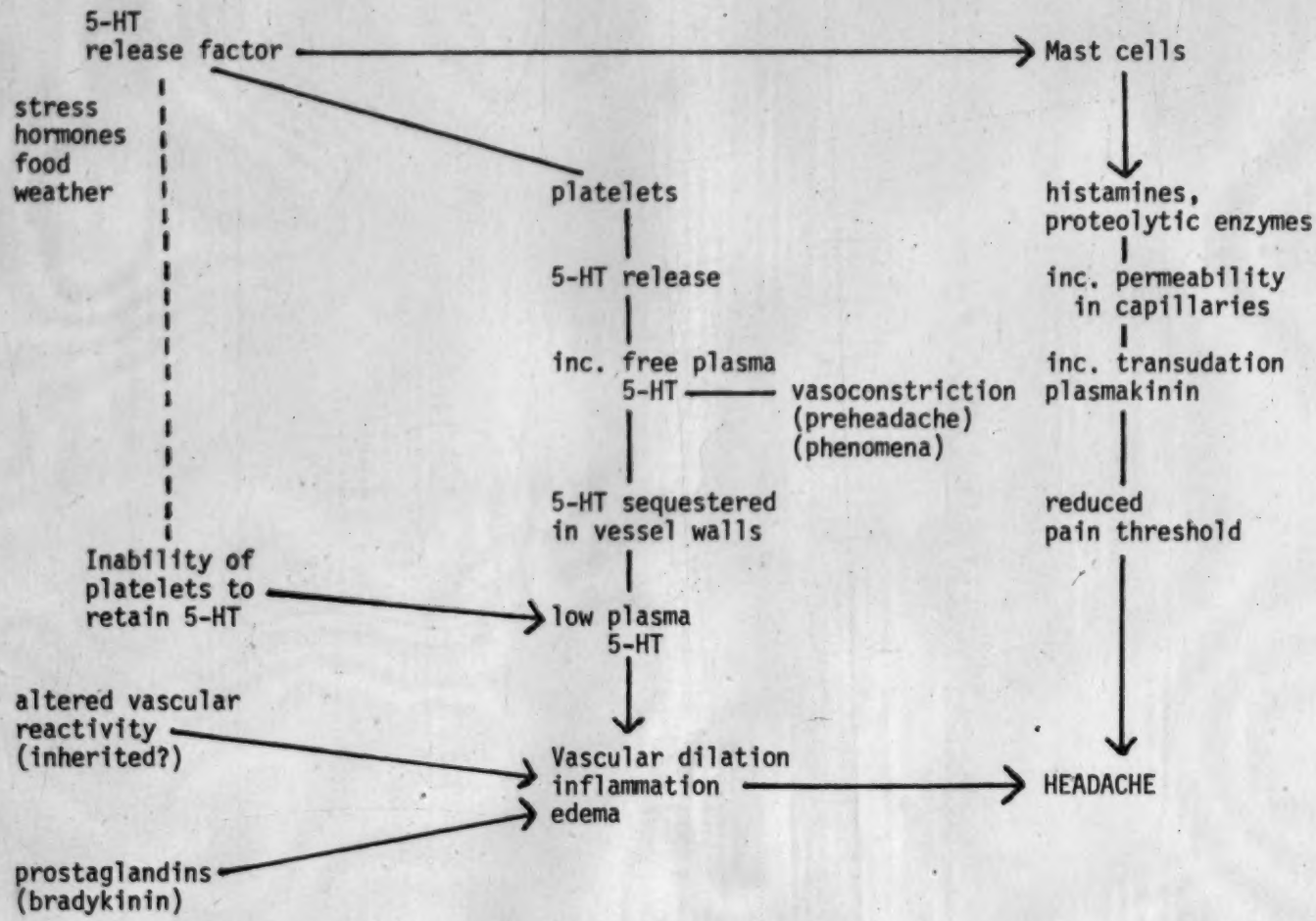


Figure 2-1

Hypothesized Biochemical and Vascular Mechanisms of a Migraine Attack

2.3 Etiology

2.3.1 External Precipitators

Vascular headaches appear to arise from a variety of factors. Certain stimuli, environmental or internal, affect the neurovascular mechanism in susceptible persons and consequently initiate a headache. A number of these external triggers have been identified.

2.3.1.1 Foods

In approximately 25% of migraine sufferers (Selby & Lance, 1960) certain foods appear to initiate a headache. Foods which have been known to induce a migraine attack include fats, fried foods, chocolate, cheeses, citrus fruits, tea and coffee, and alcohol. A number of these foods contain tyramine, which is known to be a vasoactive amine. Tyramine rich foods include strong cheeses, chocolate, pickled herring, chicken livers, canned figs and broad bean pods. Tyramine is a sympathomimetic substance which imitates the action of the sympathetic nervous system and initiates the release of serotonin from the blood platelets. Sandler (1977) suggests that an enzyme deficiency (MAO) may prevent tyramine from being metabolized, so that it is consequently absorbed into the blood stream.

Other foods observed to precipitate a migraine attack include cured meats which contain sodium nitrites, foods which contain monosodium glutamate, a known vasoactive agent, and alcohol, which is a non-specific vasodilator. Fasting, which in turn produces hypoglycemia has been shown to induce migraine attacks in susceptible persons (Rose, 1979).

2.3.1.2 Hormonal Triggers

A fall in plasma estrogen levels appears to be a precipitating factor in menstrual migraine (Somerville, 1972). Seventy percent of women report their migraine attacks to be associated with menstruation and most women sufferers are free of migraine by the third month of pregnancy (Diamond & Dalessio, 1978). Headaches may also become more frequent at puberty and there also may be a change in the headache pattern at menopause (Rose, 1978). These events suggest a connection between migraine and hormonal levels. The use of oral contraceptives and post-menopausal estrogen replacement therapies have both been demonstrated to significantly increase the frequency of migraine attacks (Kudrow, 1975).

2.3.1.2 Other Factors

Weather has been identified as a causative factor in migraine. Twenty to thirty percent of the migraine population react to (a) heat, (b) cold, or (c) thunderstorms (Sulman, 1980). Sulman identifies air electricity as a common cause in such attacks. Cold periods, blizzards and heat waves are commonly combined with a bright dazzling glare which produces positive ions and electromagnetic waves. This electrical reaction induces a serotonin release in susceptible persons who will experience a migraine reaction one to two days before the weather change since the electric phenomena travel with the speed of light, and the weather front proceeds more slowly.

Other observed precipitants of migraine include glare of flickering lights, smoking, exercise and hot baths (Rose, 1979). An association between migraine and other physiologic factors such as allergic reactions or epilepsy has been hypothesized, but there is no conclusive

evidence of any such associations (Adams, Feurstein, & Fowler, 1980), although Selby and Lance (1960) have reported abnormal EEG readings in approximately 30% of migraine patients studied.

2.3.2 Internal Susceptibility

2.3.2.1 Genetic Predisposition

Many neurologists have considered a familial history to be a prerequisite for the diagnosis of migraine (Bakal, 1975). Wolff (1963) examined the incidence of migraine in the relatives of 119 migrainous patients. Approximately 84% of these patients had at least one relative with migraine. Waters (1971) examined the familial incidence of headache in a sample of the general population. He found that, among headache sufferers, the prevalence of migraine was highest in the relatives of migraine sufferers, but this prevalence was only ten per cent. Waters concluded that too much emphasis had been placed on the hereditary basis of migraine. Lucas (1976) found a correlation rate for migraine in monozygotic twins of 26%, supporting the existence of a small genetic factor. Waters has concluded that even if migraine is more prevalent in certain families, it does not follow that this prevalence is genetically determined. Family members share a similar environment and any characteristics which occur in certain families may be the result of acquired rather than congenital disease. Whether the predisposition is genetic or acquired, one might wonder what form this predisposition might take. Sachs (1971) has suggested that the following physiological parameters might predispose an individual towards migraine -- a particular sensitivity to motion, heat, exhaustion, or depressant drugs -- exaggerated cardiovascular reflexes -- microcirculatory disorders -- or some specific metabolic or chemical dysfunction.

2.3.2.2 Personality Correlates

Two general theories have developed as to the cause of migraine headache, a constitutional model, as previously discussed, and a psychodynamic model (Schnarch & Hunter, 1979) which assumes that the migraine headaches are produced by the unconscious as a way of coping with a personality crisis. In this model the migraineur is seen to possess a 'migraineous personality', a personality type in which the person characteristically represses rage, experiences anxiety, and who is perfectionistic and obsessive. The constitutional theory and the psychodynamic theory are not necessarily exclusive. An extreme emotional reaction may be a specific trigger to initiate the migraine in a migrainous constitution.

Fromm-Reichmann (1937) was the first to put forward a psychodynamic theory to explain the migraine attack. She saw the migraine patient as one who repressed a hostility against a beloved person, a hostility which was consequently expressed in the physical symptoms of migraine. This theme of repressed anger underlies other psychodynamic theories brought forth in connection with migraine. Weber (1932) saw the migraine attack, with it's accompanying release of previously inhibited excretory functions, to be a neurotic and somatic means of finding release from mass repression. Weber related this interpretation to the strong feeling of well being which customarily follows the attacks. Touraine and Draper (1934), from their own case studies, perceived the migraineur to be typically perfectionistic, insecure, unresponsive, sensitive, anxious and frustrated, and highly intelligent. They saw the migraine attack as a syndrome comparable to other neuroses.

Wolff (1937) wrote extensively on the migraine personality, based

on a study of 46 subjects with migraine. He described these subjects as unusually ambitious, perfectionistic and efficient, inflexible persons who tended to harbour strong resentments, to be cautious and reserved, cool and detached with others. The women he observed tended to be sexually maladjusted. These people seldom attained genuine relaxation and experienced anxiety and tension in most aspects of their lives. Wolff noted the frequency of Sunday and holiday headaches, somehow related to a 'letdown' or change of pace. Wolff postulated that the individuals that he studied likely form a representative group of migraine sufferers, and that others who experience migraine might have similar personality features. Trowbridge, et al., (1934), through the use of the Bell Adjustment Inventory, verified previously quoted case study work which presents the migraine patient as psychoneurotic with health and social and emotional maladjustments. Alvarez (1947) gave support on the basis of clinical findings to the migrainous personality. He based his conclusion on a study of more than 500 migraine patients, most of whom were women. He presented the typical migrainous woman as hypersensitive, highly intelligent, one who tires easily, moves quickly, worries and sleeps poorly, a perfectionist who is easily upset. He perceived migrainous men to possess similar temperamental characteristics. Sperling (1952), a psychoanalyst, perceived migraine sufferers to be orally fixated with a depressive and impulse ridden character. She accounted for their observed rigidity, tenseness and hypersensitivity in this manner. Furmanski (1952) also with a psychoanalytic view, saw the migraine as the physiological manifestation of inhibited hostility. Kolb (1963) supported the view of the migraineur as being tense and anxious with an overgrown superego or conscience, which does not allow acceptance

of one's own inner needs and desires.

Philips (1976), using an Eysenck Personality Questionnaire on a random sample of 500 patients, did not substantiate the theory that headache sufferers are neurotic and argued that "doctors and neurologists may have formed this view of headache personality on the basis of headache sufferers who seek their help" (p. 541). Henryk-Gutt and Rees (1973) empirically examined the hypothesis that specific personality traits are predisposing factors to migraine attacks, and that stressful emotional experiences may serve as precipitating factors, using three separate psychometric tests, on a random sample of 50 men and 50 women. Their findings supported the theory that psychological stresses appear to act as important precipitants of migraine, and are in fact, a factor in 54 percent of the migraine subjects reported. Their findings also suggested that migraine subjects are predisposed to experience a greater than average reaction to a given quantity of stress. They did not confirm previous suggestions that migraineurs are excessively ambitious or obsessional. They point out that clinical studies of migraine are self-selected and not fully representative of the general population.

Schnarch and Hunter (1979) also used a questionnaire battery to examine personality characteristics of migraine sufferers and results tended to not confirm previously reported 'migrainous personality traits' and psychodynamic theories of migraine causation. The only significant difference seen in migrainous over non-migrainous subjects was a fear of expressing anger and a suspicion of other people. Pearce (1977), restated the point that the descriptions of migraine personalities are subjective impressions of selected patients. He pointed out that empirical studies tend to agree in general and indicate that the migraine

patient tends to be a neurotic personality who has difficulty in handling stress, but noted that this description is apt for persons with other disorders and also for many persons with no illness. Rees (1977) also concluded that where emotional factors and stress are important precipitants of migraine, they are not specific. He pointed out the importance of studying migraine sufferers sampled from the general population as opposed to those who attend clinics. Rees concluded that migraine is a complex disorder with multiple causative factors and that psychological stresses appear to act as important precipitants of these attacks.

In summary, a number of practitioners present the migraineur in terms of a 'migraine personality' who characteristically represses hostility, and demonstrates perfectionistic and obsessive characteristics. More empirical studies emphasize that these assessments have been based on migraine subjects who seek medical help, and that such subjects represent only a subsection of the migraine population. In one of the more important and largest of such studies, Henryk-Gutt and Rees (1973) concluded that psychological stress reactions are a factor for more than half of the migraine subjects reported, and they reiterate that migraineurs may react to a greater than average degree to a given amount of stress. It would seem, then, that stress and psychological factors are important causative factors in migraine attacks, but that these factors, while involving the characteristics of anxiety and inflexibility, are not as specific as was formerly believed.

2.3.3 The Stress Reaction

"Stress is the non-specific response of the body to any demand made on it" (Selye, 1974, p. 14). Selye elaborated that it is

immaterial whether the stressor is pleasant or unpleasant. In either case, there is a demand for adaptation or re-adjustment, and this non-specific adaption he labels the general adaptation syndrome, or the 'flight-or-fight' response. Stress is not merely nervous tension. A stressor may be any agent or situation which brings into activation the arousal mechanisms which enable the organism to adapt and maintain life. The effect of the stressor is a function of the intensity of its demand on the system, and the reactivity of the system. Selye, in 1936, through animal experiments, discovered that the same set of organ changes were induced by such diverse stimulants as cold, heat, infection, trauma hemorrhage, nervous irritation or glandular extracts. He then formulated what he called the general adaptation syndrome. Since 1936, Selye has traced the biochemical and structural changes involved in non-specific stress.

Initially the stressor may act on any part of the body and eventually produce the general stress reaction where nearly every organ and chemical constituent is involved, through the coordinating actions of the endocrine and nervous systems. Stressors act through the nerves, and through neural stimulation of the adrenal medulla to produce adrenalines and acetylcholines, two antagonistic hormones. Of particular interest in relation to migraine are the noted vasoconstrictive effects of the hormones adrenaline and noradrenaline. In stress, the endocrine system is activated through the hypothalamus which produces a substance which stimulates the pituitary to discharge the hormone ACTH (adrenocortico-tropic hormone) into the blood. ACTH in turn induces the adrenal gland to secrete corticoids which act to repress the thymicolymphatic organs and certain white blood cells which are necessary for immunity and

allergic hypersensitivity reactions. The corticoids also act on the thymus and thyroid glands, the connective tissue, the liver, and the kidneys. The kidneys are influenced in a manner which results in a constriction of blood vessels throughout the body (Selye, 1976). If the stress reaction is prolonged, a frequent reaction is the development of peptic ulcers, again produced by the increased levels of corticoids in the blood stream and by the action of the autonomic system (Selye, 1974). Paulley & Haskell (1975) report on certain similarities between migraine headache sufferers and patients with duodenal ulcers, in that they share certain compulsive traits and a high work ethic. It is of interest to note that both of these groups show a significant correlation in blood group 'O'.

Selye (1974) demonstrated that qualitatively distinct stress producing stimuli differ only in their specific actions. Their non-specific stressor effects are essentially the same, and he demonstrated further that (1) "qualitatively different stimuli of equal toxicity do not necessarily elicit the same syndrome in different people," and (2) "even the same degree of stress, inducted by the same stimulus, may produce different lesions in different individuals" (p. 33).

The fact that the same stressor can cause different lesions in different individuals he traced to 'conditioning factors' that can enhance or inhibit one or the other stress effect. Thus, conditioners may be internal (for example, genetic predisposition, age, or sex) or external (due to hormones, drugs, or dietary factors). Under the influence of these conditioning factors (which determine sensitivity) a degree of stress which normally may be well tolerated can become pathogenic and cause "diseases of adaptation", affecting certain

predisposed areas of functioning (Selye, 1974).

With this concept of conditioning Selye explains the pathology of stress. Any kind of activity may set the stress mechanism in motion, but it will depend on the conditioning factors which part of the body is first or most affected. "In the body, as in a chain, the weakest link breaks down under stress, although all parts are equally exposed to it" (Selye, 1974, p. 35).

Whatmore and Kohli (1974) defined a dysfunction of the general stress reaction as dysponesis or misplaced effort. They viewed physiopathology as an altered circuit activity within the nervous and neuromuscular systems, which in turn induces alterations in tissue and organ function. Such circuits may be inborn or conditioned. Whatmore and Kohli viewed the nervous system and endocrine system basically as complex signalling systems, systems which are subject to signal error. One significant source of signal error within the nervous system may occur in the action-potential output from the pre-motor and motor cortex, called effort or ponesis. Much of this effort is covert in nature and human beings exhibit four kinds of effort (1) bracing efforts, (2) performing efforts, (3) representation efforts, and (4) attention efforts. Errors in ponesis may occur in any of these dimensions, mostly through conditioned pathways, to produce physiologic alterations that interfere with the efficiency and health of the organism and may lead to structural change.

Whether physiopathology is defined in terms of dysponesis (Whatmore & Kohli) or as a derailment of the General Adaptation Syndrome (Selye) it would seem that for a sizeable proportion of our population, functional disorders are manifested in the form of migraine headache. The

conditioners for this reaction may be of internal or external origin.

2.4 Treatment Modalities

2.4.1 Identification and Avoidance of Triggers

If migraine is viewed as a genetically determined physiologic disposition that is influenced by a variety of emotional, biological and constitutional factors, isolating and possibly modifying these factors may be a logical first step in a treatment program. Since fasting or missing meals is a factor that may produce headache (Saper, 1978), a migraine sufferer may benefit from a regular routine of sleeping and eating. Avoidance of certain foods, especially those containing tyramine, nitrites, monosodium glutamate or alcohol, seems to be a helpful first step for those persons who are susceptible.

The use of oral contraceptives and estrogen therapy has been demonstrated to worsen migraines in susceptible women (Kudrow, 1975) and would seem to be a contraindication in such cases. In addition, there seem to be a number of miscellaneous triggering factors. Glaring sunlight or other strong light seems to provoke headache in some people. Glasses with polarized lenses may be helpful in such cases. Some migraine patients have cited physical exertion or intercourse to be precipitating factors, although exercise on a regular basis may increase an individual's overall ability to handle stress and therefore be beneficial. Reasonable consideration can also be given by the migraine sufferer to reduce the level of environmental stress factors that may be present in personal or work life.

2.4.2 Drug Therapy

Treatment may be viewed as a process of raising the body's level

of defense. The body's defense may be active or passive. In treatment with drugs, the body is passive, and the drugs act directly and specifically, rather than by strengthening the body's natural resources and when the drug therapy is discontinued, symptoms commonly reappear.

A number of analgesics, sedatives, and specific antimigraine agents have been administered by physicians to treat migraine headache. Anthony and Lance (1972) have classified the drugs which are used specifically to abort the migraine attack into four groups:

(1) those which produce extracranial vasoconstriction, such as ergotamine tartrate. Ergotamine has been demonstrated to be one of the most effective drugs in the treatment of migraine (Rose, 1979), if administered early in the attack. It is administered orally, rectally, by inhalation or by injection, and other vasoconstrictive drugs, such as caffeine may be added to increase its efficacy. Ergotamine is addictive and overdosage may worsen the headache.

(2) those drugs which simulate the action of serotonin receptor sites such as methsergide. Methsergide is an antagonist of serotonin and has been shown to be 50-65% effective in migraine treatment (Saper, 1978). This drug has strong side effects and can only be administered intermittently.

(3) those which block beta-adrenergic receptors in the blood vessels and prevent vasodilation, such as propranolol. Beta blockers block the effects of the release of adrenaline and nor-adrenaline which are in turn induced by the stress reaction. Propranolol appears to affect platelet uptake of serotonin and affect fatty acid and prostaglandin metabolism (Saper, 1978).

(4) monamine oxidase inhibitors which act to break down the

amines which act as neurotransmitters. Monoamine oxidase inhibitors have been effective in migraine treatment. This is paradoxical because MAO levels have been demonstrated to be low in migraine attacks (Sandler, 1977). These drugs have also been shown to be effective in the treatment of depression.

Drugs which are also in use, and may be classified as preventative (Rose, 1979) include, (a) analgesics, or pain killers, which include aspirin and codeine, and act centrally making the brain less responsive to pain; (b) tranquillizers and general stress relievers, such as valium and librium; (c) anti-depressants which alter nor-adrenaline levels in the blood and make people less likely to react to external stress by reducing anxiety and agitation; and (d) anti-histamines which act to prevent the inflammatory effects of histamine in the tissues.

2.4.3 Psychotherapy

Drugs may provide no lasting relief from migraine, particularly as migrainous patients tend to regard any intermission of their symptoms simply as a green light to drive themselves harder (Paulley & Haskell, 1975). Wolff also emphasized that, in the long run, individuals need to understand the factors in their lives which create tension and physical distress.

Friedman (1973) stated that "since emotional factors appear to be the most frequent precipitant of a migraine attack, treatment of the psychological problems is paramount" (p. 104). He believed, however, that the internist or neurologist should limit himself to supportive therapy, and situational guidance and counselling, and that long term intensive therapy should be done by the trained psychiatrist.

Early psychoanalysts treated migraine through extensive

psychoanalysis. In a study of 14 patients who were trained to deal with their personality problems, Wolff (1937) reported that 12 obtained long term substantial relief. Paulley and Haskell (1975) treated 800 migrainous patients over a period of 22 years and strongly agreed with Wolff that any technique will fail unless one first helps the patient to overcome deep guilt over what their superego tells them is unacceptable in terms of idleness and imperfection. Fromm-Reichmann (1937) reported that in analytic work with 8 migraine patients, 5 became essentially cured, 2 found much relief and 1 was uninfluenced. Weber (1932) presented a case study whose migrainous symptoms aborted over long term analysis. There appears to be little recent literature regarding the use of psychoanalytic techniques in migraine. Lake, Rainey, and Papsdorf (1979) included rational emotive therapy as a treatment adjunct to a group of subjects who were otherwise receiving biofeedback as a program to control migraine. The group which received RET was no more effective in controlling their headaches than those which did not. Pichel (1977) reported on a case study where transactional analysis theory and techniques of gestalt therapy were applied successfully in treatment of migraine.

In summary, the importance of psychological factors in the etiology of migraine has been recognized and documented. Such factors suggest a psychological treatment program, which in the past has been applied through psychoanalytic and psychodynamic therapeutic methods. However, these methods have produced only limited success. It would seem that while the migraineur would profit from self-understanding regarding the basis of his tensions, an intellectual understanding of psychophysiologic dysfunctioning, alone, is not sufficient to correct the dysponetic state. Specific training is required to cultivate the ability to

distinguish the functional from the dysfunctional response. Furthermore, following such training, the subject needs to understand that any benefits therein will come solely from his own continuing application.

2.4.4 Relaxation Training

Stress affects all of us. We live in a time of rapid sociological and technological change. Doctors have recognized for years that we pay a psychological and physical price in our attempt to adapt to the demands placed on us (Benson, 1975). The generalized fight-or-flight response is not necessarily appropriate in our present day situation and repeatedly elicited may lead to a number of debilitating diseases (Benson, 1975). The migraineur has a clear and striking symptom that mind and body can bear the pace no more, if its victim would but heed this message.

When the General Adaptation Syndrome (G.A.S.) or fight-or-flight response is evoked, the sympathetic nervous system is activated. When aroused, this sympathetic nervous system secretes the hormones adrenaline and norepinephrine. These hormones bring about physiologic changes of increased blood pressure, heart rate and body metabolism. The G.A.S. is controlled by an area in the brain called the hypothalamus. While this response is associated with overactivity of the sympathetic nervous system, there is another involuntary response that leads to a reduction in the activity of the same nervous system, named by Benson (1975) as the relaxation response.

This physiological state is characterized by certain bodily changes which include a decrease in the rate of metabolism, where oxygen consumption is reduced, heart and respiration rates slowed, and alpha or slow brain waves are increased in frequency. There is also a decrease in blood lactate levels, a substance related to skeletal muscle

metabolism, increased amounts of which are shown to be associated with anxiety (Benson, 1975). These physiological changes are found in persons who practice meditation, but they are not unique to meditation and in fact represent a hypometabolic or restful state.

If a relaxation state can be deliberately created, it may be hypothesized that relaxation may be used as a preventive measure in aborting migraine headache attacks, if migraine is viewed as a maladaptive aberration of the General Adaptation Syndrome. Lutker (1971) presents a case study of a migraine subject who was trained in relaxation techniques over a two week period. Symptoms were reported to be entirely gone over a two month follow-up period. Paulley and Haskell (1975), in over 22 years of treating migraineurs, developed a treatment program which included weekly relaxation training sessions. They report a success rate of 67% in their clients, observed over a 3 year follow up period. A program of relaxation training, including patient self-awareness therapy may be a means of reducing migraine attacks over a continuing period of time, and may also induce beneficial life-style consequences, without presenting any potentially harmful side effects.

2.5 Summary

Migraine is seen as a psychogenic organic disorder, manifestations of which are innervated by the autonomic nervous system. The development of migraine is a function of psychological or behavioral events which induce emotional arousal and concurrent sympathetic activity, culminating in physiological and biochemical changes, and hence 'pain'. The psychological or behavioral events are seen as primary antecedents to the syndrome. The migraine itself is viewed as secondary, being one of a number of possible manifestations of dysponesis.

Migraine treatment, in the past, has focused on two major themes, pharmacotherapy and behavior treatment programs. Neither method has so far fully alleviated the syndrome. Drug therapy exerts a temporary alleviation of symptoms which will return on the cessation of therapy. Many clients are interested in a non-drug therapy because the side effects of drug use are aversive, or costs are high, or the medication does not continue to reduce the pain. Behavioral therapies act on the primary sources of the problem, working to actively modify the dysfunctional patterns which initiate an attack, and demonstrate no harmful side effects. Behavior therapy includes psychotherapy and relaxation training. In the past, the use of psychotherapeutic and psychoanalytic techniques has demonstrated some limited success. Such programs may be time consuming and migraineurs, in general, are ill-disposed towards psychotherapy, per se. Behavior training, as initiated through relaxation therapy, as a treatment modality in itself, or combined with psychological management skills may present as a promising approach in migraine management. There is enough evidence to suggest that relaxation learned through biofeedback procedures is learned more rapidly and more effectively, and that such procedures warrant further study. As a caution, it is known that headache patients often respond to placebo effects and are apt to seek treatment when headaches are at their worst. In this event, any therapy may seem to demonstrate some effectiveness.

CHAPTER THREE

3. PROPOSED MECHANISMS OF BIOFEEDBACK TREATMENT

3.1 Overview

3.2 Biofeedback as a Behavioral Therapy

3.3 The Process of Biofeedback

3.3.1 Biofeedback as applied to temperature training

3.3.1.1 The autonomic nervous system
3.3.1.2 Voluntary control of skin temperature
3.3.1.3 Experimental applications
3.3.1.4 Summary

3.3.2 Biofeedback as applied to electromyography

3.3.2.1 The occipito-frontalis muscle
3.3.2.2 Voluntary control of muscle activity
3.3.2.3 Experimental applications
3.3.2.4 Summary

3.4 Non-Specific Learning Components (Placebo Effects)

3.5 Summary

3.6 Experimental Rationale

3.7 Research Questions

CHAPTER THREE

3. PROPOSED MECHANISM OF BIOFEEDBACK TREATMENT

3.1 Overview

Feedback is a general term, a word developed by the mathematician, Norman Weiner, and refers to a method of controlling a system by reinforcing into it the results of past performance. Biofeedback is a special form of feedback, where the system is biologic and the feedback is artificial (Birk, 1973). It should not be surprising that biofeedback may be effective since all living systems are self-regulated, and their very existence and stability depend on feedback controls, feedback being the process which creates internal homeostasis. Human beings, generally, are not conditioned to develop a sensory awareness of all the subtle feedback mechanisms in the body of which they may become aware. Through biofeedback, an electrical monitoring system detects and amplifies internal physiological processes which in the normal course of events are undetected. This information may be fed back to the subject via a visual display or an auditory tone.

3.2 Biofeedback as a behavioral therapy

Biofeedback is a behavioral control therapy, a mechanism apart from medical intrusions such as drug therapy or surgical intervention, where the locus of control is external, and the remedy is one which is applied. Indeed, in behavioral therapy, the patient is encouraged to assume responsibility for his/her own health and well being, to take an active role in learning not to be sick. In such a process, lifestyle is modified in a manner that will reduce the degree of external stressors, or the body is trained to diminish its reaction to such stressors.

Biofeedback is one means of applying such behavioral therapy to psychosomatic disorders. Biofeedback has been applied to such disorders as hypertension, cardiac dysfunction, Raynaud's disease, gastrointestinal disorders and migraine headache. Such uses are based on experimental demonstration, in animals and humans, of the achievement of voluntary control over bodily processes which are normally considered to be involuntary.

3.3 The Process of Biofeedback

3.3.1 Biofeedback as Applied to Temperature Training

3.3.1.1 The Autonomic Nervous System

In our western society, bodily functions which are mediated by the autonomic nervous system have long been considered to be involuntarily controlled, despite reports of the practitioners of yoga (Wenger & Bagchi, 1961). Only within the past twenty years has instrumental autonomic learning been demonstrated in experimental studies with animals (Miller, 1968), studies which led into experimental autonomic learning studies with humans.

The human nervous system can be divided into a central and a peripheral system. Included in the central system are the brain, the brain stem, and the spinal cord. A part of the brain which is pertinent to our interests is the hypothalamus. The hypothalamus is located in the brain, below the cerebral cortex, and acts as a coordinating mechanism between the brain and the endocrine or glandular system (Selye, 1976). The hypothalamus responds to the stressor message from the brain, (through the limbic system), and initiates the arousal process.

The peripheral nervous system innervates the periphery of the body and includes the voluntary sensory motor systems. The autonomic nervous

system is also known as the visceral or involuntary nervous system. It has parts in both the periphery and the central systems. This system maintains internal homeostasis and regulates metabolic processes such as heart rate, blood pressure, digestion, temperature maintenance and also controls the tonicity of the smooth muscles of the blood vessels. In controlling these functions, the autonomic system is chiefly regulated by the hypothalamus. Thus the hypothalamus is the body's important regulating center in maintaining visceral homeostasis and in initiating the previously mentioned fight-or-flight reaction (Jencks, 1977).

Within the autonomic nervous system are two mutually inhibiting systems, the sympathetic and the parasympathetic nervous system. These two systems innervate nearly every organ in the body. The action of the sympathetic system increases the expenditure of energy, and the parasympathetic system reduces, calms, and conserves energy. The sympathetic system acts in a general way, adjusting metabolic processes to a changing environment, to external stresses or to internal anxiety states. In acute stress, these adjustments include secretion of the hormones, adrenaline and noradrenaline, acceleration of the heart rate, increased blood pressure, diversion of blood from the skin to the trunk and the skeletal musculature, all of which constitute, in Seyle's words (1976), initiation of the General Adaptation Syndrome.

Conversely, the parasympathetic system promotes those metabolic activities which restore and maintain energy resources, including digestive processes, relaxation, blood vessel dilation, slowed heart rate, secretory and excretory processes (Jencks, 1977).

In general, in our fast paced and work-oriented society, the sympathetic system is much over-worked. Furthermore, the fight-or-flight

syndrome which served primitive man is not appropriate to our present form of social situation and will lead to increased tensions rather than problem resolution. Frequently, we deal with these stress related symptoms poorly, being conditioned to rely on external protection, rather than to develop our own inner resources.

In summary, the autonomic nervous system serves the internal organs of the body, through two mutually opposing systems, the sympathetic and the parasympathetic, whose functions are integrated to maintain internal equilibrium. Control centers for this system are primarily in the hypothalamus, although specific reactions are mediated at lower levels in the central nervous system, without involving the brain (Jencks, 1977). It would seem that in order to maintain our health, our well-being and our productivity, at our present pace of life, we need to recondition our autonomic nervous system, to induce those responses which conserve and build up energy. If our bodies can function with a decreased heart rate, a lowered blood pressure, a reduced oxygen consumption, in general, at a reduced pace, we shall experience less stress, emotional calmness and a longer life, and we have evidence that these processes can be learned. One such process, which is a component of the general stress reaction is the control of blood flow. In the fight-or-flight response, the blood pool is pulled into the trunk and skeletal muscles, and out of the periphery. It is worthy of note that the migraine sufferer commonly complains of cold hands and feet.

3.3.1.2 Voluntary Control of Skin Temperature

The psychology of learning has, in the past, separated learning into two processes, classical or respondent conditioning, which apply to functions inside the body (visceral and autonomic), and operant or

instrumental conditioning, which applies to the learning of external behavior and which is mediated by the skeletal muscles (Birk, 1973).

In classical autonomic conditioning, an unconditioned response such as salivation, will appear as a reaction to an unconditioned stimulus (food). This unconditioned response can be observed to occur on presentation of a conditioned stimulus, such as a bell, when that bell has been paired many times with the unconditioned stimulus. The instrumental paradigm of learning emphasizes a consequence or a reinforcing stimulus to an antecedent physiological response. This reinforcing stimulus can be primary (such as water available to a thirsty animal) or secondary, such as information or 'feedback' available to a human who is learning control of a previously involuntary response system. This model has been presented as the basis of biofeedback learning (Birk, 1973). The inherent weakness in this model is that the feedback as a secondary reinforcer has at no time been paired with a primary reinforcer. The model of conditional or operant learning may be an inadequate explanation of human learning in biofeedback. The feedback may operate as a reinforcer or as information. If both reinforcement and informational feedback are important in biofeedback training, comparative studies are necessary to determine the role of these two factors. An analysis of any such studies is outside the scope of our present study, although it is noted that Reinking and Kohl (1975) compared five methods of relaxation training where one method included a monetary reinforcement when a specified criterion was met. In this study the biofeedback trained groups were all successful and the monetary reinforcement did not increase the effect, and Sachs (1976) concluded that in biofeedback, reinforcement alone does not produce behavioral change, but that the role

of reinforcement is to increase motivation, rather than to directly effect the learning of the response.

If the learning that occurs in the biofeedback assisted situation is not conditioned or operant learning, and if the actual feedback is not reinforcement but is classified as information or motivation, the question is raised as to what does precisely occur. Yates (1980) presents a 'servomechanical model' of biofeedback control, a model which looks at the complex and refined 'feedback control' systems in the body which function below thresholds of awareness, only reaching levels of awareness when some dysfunction occurs. An example of such a feedback loop is seen in normal muscle activity, where controlled and smooth action is attained without conscious control or direction. In muscle action, the alpha motor neurons which initiate a muscle contraction have connections with gamma motor neurons which initiate the stretch reflex. It is important to note that in this process, control can be in the spinal cord, or at different levels of the nervous system. Four hierarchical levels of control are postulated (Harris, 1971) to operate in the spinal cord, the brainstem, the cerebellum, and the cerebral cortex. Afferent (incoming) impulses may travel to each level, but efferent (outgoing) impulses may or may not be released. The effect of training may be to release efferent impulses from such a center.

In the feedback training situation, the subject is provided with a feedback display (information) which reflects changes in a designated function, changes which are normally initiated at a level below the cerebral cortex, or below conscious awareness. The subject is required to increase his voluntary control over this function utilizing the feedback which is not normally as specifically available. In time this

control becomes spontaneous and can be exercised without the explicit display. The subject is unable to describe in words how this control is exercised because the message is not a step-by-step directional control but a non-verbal communication between unconscious neuro control centers (Yates, 1980).

In summary, biofeedback is viewed not as an operant conditioning process where the biofeedback display is seen as a reinforcer to a desired stimulus, but primarily as a means of providing cognitive information regarding an internal feedback loop which is normally self-correcting at a control center below the cerebral cortex. It is postulated that in this normally self-correcting system, when an error signal reaches a certain criterion, correction at a higher center is necessary. With practice the corrective response is habituated, the feedback display is no longer necessary, the feedback system is again internalized. This theory may account for the specificity or general effect that biofeedback training may serve, depending upon the level of the control center where the correction is made. The autonomic nervous system is seen to be reactive to stress inducing stimuli, whether such stimuli be physiologic or emotional. In migraine, the process is dysfunctional, whether through an overreactive stress response, or as a result of an overly labile vascular system. Biofeedback is introduced as an informational or motivational behavioral training agent whereby hand warming serves as a target response in initiating a decreased sympathetic outflow from the autonomic system. As a specific response mechanism, hand warming with concurrent peripheral blood volume changes may be seen to reduce cranial blood flow, and hence exhibit an effect in reducing the painful vasodilation phase of the headache. The criterion response is

hand-warming, which is associated with the presumptive root problem, excessive sympathetic outflow. To this end, the recording instrument is a thermistor, which measures body temperature, applied to the finger. The feedback is achieved by reading a dial which swings to the right as the hands warm, or through an auditory tone which changes in pitch in response to temperature changes.

3.3.1.3 Experimental Applications

The use of hand temperature training as a physiological response mechanism employed in a therapeutic approach to migraine headache is based on a chance observation by Sargent, Green and Walters (1972) at the Meninger Clinic, of a subject who increased her hand blood flow and experienced a spontaneous migraine recovery. In subsequent studies, (Sargent et al., 1973), this performance was verified, using a combination of biofeedback and autogenic training. Wickramasekera (1973) confirmed Sargent's report in two case studies where temperature training was implemented. Wickramasekera attributed his positive results to a change in sympathetic nervous system function, rather than a specific change in blood flow. His conclusions were that biofeedback involved aspects of operant conditioning and skill learning.

Johnson and Turin (1975) reported positive results with one individual who was trained to lower and then to raise her peripheral skin temperature. They reported that migraine activity increased during cooling and decreased during warming. The subject was given expectations for improvement during both cooling and warming conditions. Johnson & Turin interpreted their results in terms of a specific relationship to vasodilation and constriction in migraine.

Andreychuk and Skriver (1975) compared the effectiveness of hypnosis,

hand warming training, alpha wave enhancement, and suggestibility in headache management. Results showed reduced headache activity in all groups with no one treatment superior to the others, although highly susceptible individuals responded better to all treatments. Such results would indicate a general training effect on dysponesis rather than any specific symptom alleviation.

Taub and Emurian (1976) reported further positive results in training subjects to control skin temperature, and thereby to control the volume of blood flow in peripheral vascular beds. In their study, the magnitude of change varied with sex, direction of change, and increased with the use of performance rewards.

Surwit, Shapiro and Feld (1976) demonstrated success in training subjects to achieve bidirectional control over digital skin temperature. Sex differences were seen in response to voluntary vasodilation. As females warmed, their heart rates increased. As males warmed, their finger temperatures, heart rates were seen to decrease, suggesting that a different process was involved in males and females.

Daniels (1976) administered hypnotic instruction and hand warming instructions by tape, and found these methods to be effective in reducing frequency, intensity and duration of migraine attacks in four subjects. All subjects reported that they commonly overreacted to stress, previous to the training program.

Turin and Johnson (1976) studied the effects of biofeedback training in migraine headache. Migraine was seen to decrease as a function of hand warming, and to show no improvement in conjunction with hand cooling. Subjects were trained to cool before warming, and no autogenic training was included. Such results mitigate against a placebo or

expectancy effect.

Instead of a temperature reduction procedure, Friat and Beatty (1976) trained migraine suffers to decrease pulse amplitude, in the forehead or peripherally. They reasoned that head pulse amplitude should be specific to migraine symptoms and hand pulse amplitude related more generally to the syndrome. Results indicated that only those subjects who demonstrated vasoconstriction in the extracranial arteries experienced improvement in headache symptomology.

Blanchard et al. (1978) assigned 30 patients to three experimental groups. One group received temperature biofeedback and autogenic training. Another group was instructed in progressive relaxation. Each of these groups was instructed to practice regularly. A third group was assigned as a waiting list control. After a follow-up period, both treatment groups demonstrated a significant and equal improvement in headache activity. Blanchard and his associates concluded that their data provided no basis to choose one treatment over the other, and that both treatments operated through the common pathway of relaxation.

Silver et al. (1979) reported similar results. In a comparison of temperature biofeedback and progressive relaxation training in treating migraine headaches, gains achieved in either program were equal at the end of a one year follow-up period. Conclusions again were that both treatments may be acting through a process of general relaxation and a reduction of sympathetic arousal.

3.3.4 Summary

On the basis of recent studies it would appear that voluntary digital skin temperature control is achieved relatively easily, and that

such control leads to a reduction in migraine activity. Many of these studies included autogenic relaxation training. Conclusions seem to be generally that the therapeutic action gained through finger temperature training is due to a general reduction of sympathetic outflow. While biofeedback training in digital vasodilation has been shown to be effective, it has not been proven to be more effective than relaxation training. However, in the development of self-control procedures for the treatment of migraine, biofeedback is seen as a striking sensory based method of inducing the relaxation response and making the effects of such a response visible and evident to the client, rapidly and effectively.

3.3.2 Biofeedback as Applied to Electromyography

3.3.2.1 The Occipito-frontalis Muscle

The occipito-frontalis is a broad striated musculo-fibrous layer which covers the whole of the crown of the skull from the occiput to the eyebrow. The frontal portion, called the frontalis muscle, has long fibres which are blended with other facial and neck muscles (Gray, 1975). Electrodes then, when placed on the frontalis muscle register muscular tension throughout much of the head and neck.

Striated muscle is made up of bundles of fibres, which may be up to 30 cm. in length, but in width less than 0.1 mm. These fibres can contract or shorten by up to 55% of their resting length. In a particular muscle, the fibres do not shorten simultaneously but successively, to an extent depending on the degree of neural stimulation. Because these successive contractions occur in very large numbers of fibre bundles, the muscle appears to contract smoothly as a whole. Any group of muscle fibres which contract together are innervated by an

electrical impulse from a single neuron. The impulse travels from the cell body of the neuron through the axon or nerve fibre and through nerve endings into groups of muscle fibres. This single motor unit (SMU) is the fundamental building block of muscle activity. The number of fibres which are innervated by a single neuron varies from a small number in fine muscle function to a large number when the muscle action is more gross. When a muscle is activated, the smaller motor units are recruited first, with large units becoming activated as the stimulation increases. As the number of activated units increases, the frequency of firing of each unit also increases. It is possible to record muscle activity because the contraction of each muscle fibre generates a brief and minute electrical impulse. Each impulse may endure for approximately 10 msec, and have an amplitude of only millionths of a volt (Yates, 1980).

This electric potential may be detected by deep or surface electrodes. The minute signal is detected and distinguished from surrounding noise. Because the signal is so small, amplifications of up to 100,000 may be required.

3.3.2.2 Voluntary Control of Muscle Activity

Voluntary control of striated muscle has been demonstrated by Budzynski and Stoyva (1970) on the frontalis, and by Green et al.(1969) on the forearm muscle. Each study demonstrated that voluntary control of striated muscle becomes possible when visual or auditory feedback of the muscle activity is presented to the subject. These demonstrations have involved determining the resting level of the muscle without feedback, and then providing feedback with instructions to the subject to lower the level of muscle activity. A reduction of muscle activity has been of specific interest in the training of subjects who present

abnormally high resting levels, such as are seen in tension and in migraine headaches. Biofeedback was applied to tension headache by Budzynski, Stoyva, Adler and Mullaney (1973), who stated that patients in their study exhibited frontalis muscle resting levels above normal levels and that EMG training reduced these levels and the incidence of headache. Budzynski and his colleagues reported further that these subjects showed improvement in respect to levels of depression, tension, anxiety, insomnia, and drug usage, indicating a change in sympathetic and parasympathetic functioning. The question is asked, as to whether EMG biofeedback training is effective through a specific action on one set of muscles or does it act through the initiation of a general relaxation response. Since Budzynski's (1970) initial pilot study and his later work (1973), evidence has been gathered to support either or both of these explanations.

In relating EMG training to migraine, there is evidence (Bakal & Kaganov, 1977) to suggest that frontalis muscle activity levels are as high or even higher in migraine than in tension headaches, both in the resting state and while a headache is being experienced. There is also evidence that training in the voluntary control of a specific function, not necessarily related to the presenting disorder may have beneficial effects on that disorder (Yates, 1980). Such diverse conditions as asthma, alcoholism, tension headache and anxiety have responded positively to frontalis muscle relaxation training.

3.3.2.3 Experimental Applications

Following the work of Budzynski et al. (1970, 1973), Reinking and Kohl (1973) studied the relative effectiveness of four types of relaxation

training. All of the experimental groups reported increased relaxation, but in speed of learning and depth of relaxation EMG groups were superior. A control group demonstrated no mastery of relaxation skills. Reinking & Kohl concluded that only the biofeedback procedures work well in producing high relaxation levels in a short period of time. Reinking & Kohl suggested, however, that cognitive set and personality variables may also affect relaxation skill acquisition.

Cox, Freundlich, and Meyer (1975) compared EMG training and progressive relaxation training administered to experimental groups of tension headache sufferers. Comparisons of follow-up data indicated that biofeedback and relaxation training were equally effective in the reduction of pain. Cox suggests that optimal results may be obtained by combining biofeedback techniques and relaxation training.

Paulley and Haskell (1975) combined relaxation with psychological management techniques in the treatment of migraine. Two out of three subjects demonstrated major improvement in headache activity. Paulley & Haskell recognize thatmigraine sufferers typically are initially alarmed at the idea of relaxation and that relaxation techniques will fail unless subjects are helped to overcome their guilt feelings as related to idleness. Paulley & Haskell's program did not include biofeedback training, but is included here because of the general contribution of their study to migraine and relaxation.

Schandler and Grings (1976) found muscle relaxation treatment, and combined relaxation and biofeedback training to be more effective than biofeedback alone in muscle contraction headaches, indicating that relaxation training and practice are the essentials in the treatment.

Sturgis, Tollison and Adams (1978) applied EMG feedback and blood

volume pulse feedback (BVP) separately to two subjects who suffered from combined migraine-muscle contraction headaches. In each subject, application of the EMG training reduced the muscle contraction headache, and application of the BVP training reduced the incidence of the migraine headache. The results of this study indicate the existence of two separate pain mechanisms and of specific treatment mechanisms in the elimination of these types of head pain. Lake, Rainey and Papsdorf (1979) compared electromyographic training, digital temperature feedback and digital temperature feedback plus rational emotive therapy in treatment of migraine clients. EMG trained subjects were most effectively trained in reducing their headaches. Relaxation training was included in the biofeedback therapy. Lake and his associates concluded that EMG training is easier to learn, as compared to temperature training, and that subjects were able to meet performance criteria easier.

Bild and Adams (1980) compared the specific therapeutic effects of blood volume pulse and electromyographic biofeedback on migraine headaches. Their results indicated that while both methods were effective, EMG feedback was effective to a lesser degree than BVP feedback. These results may indicate a specific treatment effect where the pain may be primarily the result of vascular mechanisms.

3.3.2.4 Summary

In conclusion, EMG frontalis feedback has been demonstrated to be at least as effective as relaxation training, alone or in combination, in reducing specific muscle tension, and in producing subjective reports of relaxation. The physiological rationale for the application of frontalis EMG feedback as a means of achieving general relaxation is not entirely clear, and more research needs to be done on the effects of EMG

feedback assisted training on the activity of a variety of muscle groups (Surwit & Keefe, 1978). In addition, it is possible that individual differences may affect the effectiveness of EMG training when used as a method of general relaxation.

The use of EMG frontalis training may then be investigated in migraine headache on the basis of specific pain alleviation in the case of accompanying tension pain, and through the initiation of a general relaxation effect.

3.4 Non-Specific Learning Components (Placebo Effect)

A headache is more than a physiological mechanism, an EMG reading, or a measurable increase in vasodilation. A headache has psychological components. Psychological factors are involved in the initiation of the headache, in accommodation to the pain mechanism, and in responses to therapeutic programs. Such affects have not been commonly controlled for in biofeedback studies, but are mentioned here as possible confounding factors.

Personality, age, and experimenter attitudes are cited as factors most apt to influence experimental results. Sargent et al. (1973) found that younger persons responded more quickly to training. He saw younger persons as being less rigid and more ready to adjust to new situations.

Personality factors cited include attitude and motivation. A successful attitude for effective biofeedback induced control is one of passive attention. Taub and Emurian (1976) noted that when subjects tried too hard, results were counterproductive, but when subjects relaxed, and simply focused their attention on the available feedback, success was

often achieved. This successful attitude has been termed 'passive volition' (Sargent, Green, & Walters, 1972). Contrarily, a lack of faith or belief in the treatment can cause a subject to assess a lack of immediate success as failure, and thus to 'give up'. Sargent et al. (1973) also noted the importance of psychological factors in hand warming, and believed that people comfortable with the hypothesis that thoughts and feelings can influence bodily processes learn faster.

Taub and Emurian (1976) report that no MMPI scales correlate with an ability to alter skin temperature, but that performers do report feelings of relaxation in conjunction with successful performance of this task.

Budzynski (1973) believed that biofeedback is more effective in conjunction with psychotherapy, as problems in the environment may prevent patient progress. The patient may be receiving reinforcement from family and friends in maintaining the headache, or may be using its symptoms to avoid difficult situations.

Locus of control, as a personality dimension, appears to be a factor in biofeedback performance. The construct of locus of control was originated by Rotter (1966) and refers to differences in an individual's attitudes towards control. Those who believe that they exercise control over their own lives are distinguished from those who believe that fate or outside events determine their lives. As biofeedback involves a learned control of internal processes through external cues, this construct may be useful in differentiating biofeedback learning ability. Carlson (1977) compared individuals who scored extremely high as internals or externals on a locus of control scale in an EMG training program. All groups who received feedback reduced their EMG levels, with the high internal scoring group attaining the lowest EMG readings.

He hypothesized that these persons may be more active in attempts to deal with their environment, although Purdine and Napoli (1971) found that exhibitionistic individuals who were more directly oriented to external sources of reinforcement (encouragement or reactions from others) performed more successfully in a study which involved biofeedback assisted control of heart rate. Cummings and Trabin (1980), using Rotter's internal-external dichotomy in separating subjects in a relaxation and biofeedback training program found that externals preferred biofeedback training, and that internals chose relaxation training. They concluded that externals responded to the external suggestion and experimenter attention.

Wickramesekera (1973) found a correlation between biofeedback skill and hypnotizability. He noted that the self-disciplined, highly motivated, less resistant person was more receptive to biofeedback.

Experimenter effects are also seen to be significant in biofeedback training. Keefe (1975) reported that the instructions given by the examiner influenced individual ability to learn temperature control. Hicks (1970), in a study of experimenter effects on a physiological experiment, randomly assigned eighteen subjects to three experimenters, who were (1) reserved, (2) automated, and (3) social. The subjects were presented with taboo words and acceptable social alternatives tachistoscopically. Subject word detection accuracy was significantly dependent on the examiner, as was their physiological response. Subjects did not verbalize taboo words accurately to the reserved or automated experimenters, and heart rate increases and vasoconstriction accompanied these inaccurate responses. Hicks concluded that such effects may largely account for discrepancies in psychological research and in human

physiological experiments.

Meichenbaum (1976) believes that the process of biofeedback depends on client cognitions, which may be in part determined by the experimenter. Meichenbaum theorizes that a shift in cognitions can mediate a shift in autonomic functioning. The trained experimenter can initiate a change in attitude from a 'learned helplessness' where certain cues foretell the onset of a headache to a 'learned resourcefulness' where a reinterpretation of these same cues can be used to initiate the responses that will abort the headache.

In summary, the placebo effect is seen to be a very real but unspecified effect due to psychological rather than physiological or biochemical variables. Such effects are focused primarily in two areas, and include client personality factors which involve motivation and locus of control (external versus internal), and experimenter attitudes where the informal, friendly experimenter who teaches the client to "reframe" helplessness into resourcefulness may greatly facilitate learning.

3.5 Summary

Biofeedback is seen to be a behavioral therapy, not where a 'cure' is administered, but a process where the patient learns to control his own dysfunctional physiological responses. The underlying theory of biofeedback assisted learning has been examined with an emphasis on informational rather than conditioning aspects. Biofeedback has been examined as applied to the migraine headache syndrome in two separate treatment modalities. Digital finger temperature training is examined as a means to achieve voluntary control over the physiological process of peripheral vasodilation, a process which is normally mediated through the autonomic nervous system, and is associated with general arousal and

relaxation responses. Electromyographic training is examined as applied to the occipito-frontalis muscle. Tension in this muscle is associated with headache pain of both the migraine and tension type, a tension which can be reduced with biofeedback assisted sensory awareness. Current experimental studies of both these therapeutic methods have been examined. The powerful effects of psychological factors in biofeedback learning are included in our assessments of the learning process.

Experimental Rationale

In this study the etiology of migraine has been examined in a search for the primary mechanisms underlying the condition, the rationale for such a search based on the belief that effective therapeutic intervention will touch those processes basic to the disorder.

Our conclusions have been that migraine has a psychogenic basis, a primary factor being the subject's dysfunctional reaction to environmental stressors. Such a premise seems to indicate a behavior therapy program. Migraineurs, who are typically of a somewhat rigid and inflexible nature are not readily amenable to psychotherapy or relaxation training. In contrast, subjects may accept the 'impressive' biofeedback instrument as a scientific advance that will effect a 'cure'. Biofeedback has been demonstrated to produce specific training effects and to facilitate general relaxation. On this basis, the use of biofeedback instrumentation is presented as an efficacious means of introducing relaxation training. With the instrument, the client can 'see' concrete evidence of the results of his efforts at relaxation. Through daily practice of such relaxation, his general tension and anxiety levels are lowered, and in addition, a specific skill is learned that may be applied when warning signs indicate the onset of a headache.

Research Questions

The goal of this study is to explore a procedure for biofeedback and relaxation training which will maximize pain reducing effects. Questions relate to the most effective means by which biofeedback training may be applied, and what variables affect this process. Two biofeedback methods are under study, EMG plus progressive muscle relaxation training, and skin temperature plus autogenic training. To further intensify the effects of such training, subjects are divided into EMG and temperature reactive groupings. Of interest, secondly, is the question of duration and extent of training. The effect of setting a pre-determined criterion and training the subject to realize this criterion, in either mode of training is examined. Thirdly, the action of unspecified variables in headache reduction is questioned, and these variables are examined anecdotally.

The basic research question of this study is then: What are the effects of a biofeedback and relaxation training program on migraine headache and what factors are involved in this process?

Specifically,

1. What are the effects of hand temperature training versus frontalis electromyographic control on headache activity?
2. What is the relationship between the attainment of a pre-determined criterion and headache reduction?
3. What non-specified variables in the study may influence subject headache reduction?

CHAPTER FOUR

4. METHOD

4.1 Subjects

4.2 Apparatus & Facilities

4.3 Research Design

4.4 Procedures

4.4.1 Stress Profile Procedure

4.4.1.1 Differential Recovery Calculations

4.4.2 Treatment Procedure

4.4.2.1 Group I: EMG Training
4.4.2.2 Group II: Skin Temperature Training

4.4.3 Follow-up Procedure

CHAPTER FOUR

4. METHOD

4.1 Subjects

The experiment included subjects who suffered from migraine headache, with an incidence which ranged from once a month to daily. Subjects responded to television and newspaper announcements of the study and its purposes. The announcements included an invitation for migraine sufferers to volunteer as subjects. Respondents were given an initial screening by telephone in response to their request for further information and entry into the program. The screening procedure (Adams, Feurstein & Fowler, 1980) was designed to insure that the subjects suffered from headaches which were indeed of the migraine type. This procedure required that three of the following criteria be met; (a) the headache was unilateral, (b) the pain was pulsating, (c) the subject experienced sensitivity to light during the headache period, (d) the headache was accompanied by nausea or vomiting, (e) the headache was diagnosed as migraine by a physician. Subjects were excluded who experienced headaches less than once a month.

In addition, subjects were excluded if they were on hormonal or birth control medication, were pregnant or were receiving psychotherapy, as such occurrences may have obscured results. A sample of the headache questionnaire employed in the screening process is shown in Appendix A. Following this initial screening, interested and qualifying persons were invited to an introductory meeting where information was shared by the three researchers clarifying the nature of the study, the rationale underlying biofeedback and relaxation

training, and the cost of participation which was a total of $50.00. Expectations regarding the subjects' participation in the study were also outlined. These expectations included daily monitoring of headache activity, regular attendance at training sessions, and daily relaxation practice. Following the decision to participate, the subject and one of the researchers signed a written contract (Appendix B) whereby the subject agreed to meet the commitments of the study and the experimenters agreed to keep personal information confidential. In addition, the researchers required that a medical certificate (Appendix C) be signed by the subject's physician, verifying the diagnosis of migraine and indicating his or her knowledge of and permission to participate in the study. Subjects completed a further questionnaire which elicited more detailed information regarding headache symptomology and general stress reactions (Table 4-1). After all requirements were met, at the commencement of the study, 68 clients were prepared to begin training. Throughout the course of the study, 14 subjects dropped out or failed to complete headache data forms, leaving a total of 54 participants. For purposes of analysis, a further 6 subjects were excluded, to equalize distributions by cell. Forty-eight subjects remained, including 42 women and 6 men, who ranged in age from 20 to 59 years. The median age was 38. Each subject had experienced severe headaches for a minimum period of two years.

4.2 Apparatus & Facilities

Biofeedback sessions took place in a 17 ft. by 10 ft. laboratory. The room was softly lit by two 100 watt light bulbs. Subjects were seated on a comfortable lounge chair facing a table containing the

Table 4-1

Characteristics of Study Population (N=54)

		Number Answered True
1.	Able to tell that a migraine is coming before the headache actually begins.	32
2.	Able to tell that a migraine is coming through visual changes or distortions.	23
3.	The head pain frequently exists on one side of the head only.	49
4.	The head pain usually exists in the temporal regions (at eye level on the side of the head).	38
5.	The head pain usually exists in the forehead region, between the eyebrows and hairline.	23
6.	The head pain usually begins in the neck at the base of the head and then radiates toward the temporal and forehead regions.	21
7.	The headaches occur in many different regions from time to time.	16
8.	The head pain usually occurs in the region at the top of the head.	12
9.	Frequently the headaches are throbbing, pulsating headaches.	47
10.	The headaches are usually characterized by pressure on the head, the sensation of which might be described as a tight band across the forehead and around the head.	28
11.	The headaches usually only occur during menses.	5
12.	The headaches occur during menses and at many other times.	42
13.	Nausea or vomiting generally accompany the headaches.	43
14.	Sensitivity to light generally accompanies the headaches.	51
15.	Sensitivity to sound generally accompanies the headaches.	52

Table 4-1

... continued

		Number Answered True
16.	Tears and nasal stuffiness generally accompany the headaches.	19

When confronting a stressful situation, the following reactions are experienced:

17.	oily skin	5
18.	sweaty feet	14
19.	flushed face	41
20.	frequent need to urinate	29
21.	cold hands	33
22.	burping	0
23.	face feels hot	42
24.	tight stomache muscles	37
25.	sweaty hands	38
26.	gasiness	12
27.	acid stomach	17
28.	shallow, rapid breathing	25
29.	cold feet	25
30.	diarrhea	10
31.	palpitation	31
32.	short breath	24
33.	shaky hands	32

biofeedback equipment. The data acquisition equipment was on a separate table accessible to the experimenter, who was present in the room throughout the session. EMG readings were obtained from standard silver-silver chloride type electrodes placed on the frontalis one inch above each eyebrow and four inches apart with a reference electrode in the center, a procedure developed by Budzynski et al. (1973). The electrodes were connected to an Autogenic Systems Incorporated 1700 electromyograph set through a shielded cable.

Impedence levels of less than 10,000 ohms were maintained throughout, as recommended by the manufacturer. The EMG signal was processed through a 100-200 Hz frequency bandpass using a one second response averaging mode. Biofeedback was provided to the subject visually on a meter gauge which displayed the EMG level in microvolts, and by a variable frequency auditory clicking through a set of Koss stereo headphones.

Finger temperature readings were obtained with a research grade thermistor attached by tape to the middle phalange of the middle finger of the non-dominant hand (Surwit et al., 1976). The thermocouple was connected to an Autogenic Systems Incorporated 2000b temperature monitor or an Autogenic HT-2. Temperature feedback was provided visually to the subject via a meter gauge indicating fahrenheit degrees and a variable auditory tone, again relayed through Koss stereo headphones. The pitch of the auditory tone was inversely proportional to changes in temperature.

The feedback myograph and thermometer were connected to optically isolated A/D converters, which in turn were processed simultaneously through the data acquisition center, which was calibrated to provide a

micro-volt/second integrated voltage value. The average level of response over 10 seconds of each minute was recorded by the printer unit during treatment sessions.

4.3 Research Design

The research design was a two (treatments) by two (groups) repeated measures across treatment phases format. Four general introductory meetings were held in the month of April and subjects were asked to monitor headaches from this point on. A three week baseline period was allowed before treatment sessions were scheduled to begin, to provide some measures of pre-headache activity. During this baseline period, all subjects were seen for a psychophysiological stress profile session. On the basis of this profile, subjects were divided into two main groups by a median split procedure. Subjects were categorized according to physiological recovery from stress into two groups, those who were most highly EMG reactive and those who were most highly temperature reactive. One half of the subjects whose stress profiles demonstrated relatively long periods of EMG reactivity with relatively short temperature reactivity were assigned to an EMG training program, the remaining half of the subjects who demonstrated relatively long periods of EMG reactivity with relatively short periods of temperature reactivity were assigned to a temperature training program. Similarly, the subjects whose stress profiles demonstrated long periods of temperature reactivity were divided into two groups. Since all stress profiles were not completed when the first treatment sessions began, the first twenty-four subjects were randomly assigned. Further assignments to groups were made ensuring that the above specifications were met.

The treatment by groups arrangement is shown as follows:

	EMG	TEMP
High EMG Low temperature reactive	12	12
High temperature Low EMG reactive	12	12

Treatment sessions ranged from four to twelve, depending on when a pre-set criterion was reached, and were carried out through the months of April through to July. The physchophysiological stress profile was repeated in a follow-up session approximately four weeks after the cessation of treatment. Thus, comparative data regarding pre- and post-treatment EMG and temperature levels during relaxation, stress and recovery periods were provided.

4.4 Procedure

4.4.1 Stress Profile Procedure

Psychophysiological stress profiles were conducted to measure relaxation, stress reactivity and recovery rates in the dimensions of digital skin temperature changes and EMG levels. First the subject was instructed to relax with eyes open for a 5 minute adaptation period, during which time EMG and temperature levels were recorded and the experimenter assessed that all equipment and monitoring systems were recording properly. Quiet background music was provided to enhance relaxation. At the end of this period the subject was instructed to continue relaxing deeply for a 15 minute period, only now with eyes closed. Following this 15 minute period a period of stress was initiated in which the subject was instructed to subtract successive sevens from one thousand, as fast as possible, for a 3 minute period. This was performed silently and with eyes closed. To

increase the stress inherent in this task, the subject was told in advance that the experimenter would ask for and record the final answer. Finally a 5 minute recovery period was conducted, during which time the subject was again instructed to relax with eyes closed. No feedback was given to the subject at any time during the stress profile session. (Appendix D)

4.4.1.1 Differential Recovery Calculations

EMG and temperature levels were monitored every 20 seconds. Stress reactivity was calculated according to a procedure developed by Carney (1981). EMG reactivity was calculated by determining the recovery time in seconds following the application of a stress, to return to the mean EMG level obtained during the last minute of relaxation. If readings during the first minute of stress did not exceed the relaxation mean, scores obtained were considered to indicate no reactivity and were assigned a value of 20 seconds. If the subject did not recover from stress at any time, recovery was counted as 500 seconds (maximum time allowed).

Temperature reactivity was calculated by subtracting the lowest temperature value obtained during the stress or recovery period, from the final temperature reading during the 15 minute relaxation period, and recording the recovery time to 50% of the drop. As was the case with EMG, negative or null temperature reactivity scores were considered to indicate no reactivity, and were assigned the value of 20 seconds, and non recovery was recorded as 500 seconds. Temperature and EMG reactivity values were converted into z-scores, and hence into T-scores. Temperature T-scores were then subtracted from EMG T-scores and subjects were divided into two groups by a median split procedure.

These two groups, with (a) relatively high EMG reactivity, or (b) relatively high temperature reactivity constituted the groups used for analysis in the experimental research design.

4.4.2 Treatment Procedure

Subjects in both treatment conditions recorded daily headache intensity, frequency, and medication consumption during the three phases of the study, including the pre-treatment, treatment and follow-up periods, using a rating scale developed by Budzynski, Stoyva, Adler and Mullaney (1973). On the forms provided the subject was asked to rate headache activity for each waking hour of the day on a scale from "0" for no headache to "5" for an intense incapacitating headache. The forms included seven daily grids to simplify recording procedures, an explanation of the severity sale, (Appendix E), and were collected weekly as completed.

Subjects were treated in pairs, twice weekly for a period of from two to six weeks. EMG subjects were given unilateral training and temperature trained subjects received bi-lateral training. The experimenters were three graduate students in Educational Psychology with experience in counselling and in biofeedback. The experimenters worked with individual subjects on a random basis, throughout all periods of the study.

4.4.2.1 Group I: EMG Training

Subjects were trained to a pre-set criterion, which was the attainment of all EMG readings below 1.5 microvolts for two consecutive training sessions. If subjects did not achieve this criterion, twelve training sessions were administered. If criterion was reached

at any session prior to the twelfth and after the second session, weaning procedures were begun. To wean, subjects underwent a 5 minute session with feedback, followed by a 5 minute session with no feedback (times 2). When the subject was able to maintain EMG readings below 1.5 microvolts during all 4 sessions, weaning procedures were completed, and the subject was considered to no longer need active biofeedback in order to achieve deep levels of relaxation. With this procedure any given subject received a minimum of 4 training sessions or a maximum of 12. Within each treatment session, EMG trained subjects experienced a 5 minute adaptation period, followed by a 2 minute baseline, 2 minutes with instructions to reduce muscular tension with no feedback, and 3 training sessions of 5 minutes each, where feedback was provided, and instructions were given to relax. A 1 minute rest period was provided between training sessions. Subjects were presented with a rationale statement (Appendices F & G) to be read on the first, the fourth and the eighth treatment sessions. The rationale statement included stages of biofeedback guided relaxation and some strategies as to how to achieve a passive attitude. In addition, therapists regularly reiterated the treatment rationale and initiated suggestions regarding strategies that may prove helpful to the subject in training. EMG training procedures are described in Appendix H.

At the end of each session, subjects were asked to spend 5 minutes writing down a description of the strategies which they had used successfully and to record any feelings or sensations which they experienced during the session as they relaxed. At the end of each session, subjects were shown their results and were given reinforcement

regarding their progress. On the initial session subjects were given a relaxation tape based on Jacobsonian (1933) progressive muscular relaxation techniques and recorded by Dr. G. Fitzsimmons. Subjects were instructed to use this tape daily during the treatment period, incorporating relaxation strategies which had been successful in the biofeedback training period. Subjects were also instructed to employ the cognitive strategies which they had found successful in the lab during their daily relaxation period throughout the day, and especially at any time during the day when a stressful situation was experienced.

4.4.2.2 Group 11: Skin Temperature Training

Laboratory training for the temperature trained group was similar to that given to group one with the following exceptions. On the initial training period, subjects were given an autogenic relaxation tape based on Schultz and Luthe's (1969) autogenic training techniques, again recorded by Dr. Fitzsimmons, plus a 'Biotic Band' digit indicator, with instructions for these materials to be used on a daily basis. The 'Biotic Band' recorded temperature changes from 78°F to 96°F, and were obtained through Biofeedback and Stress Management Publishing, Seattle, U.S.A. They provided the subject with a certain degree of feedback, even when away from the biofeedback machine. Instructions for use are included in Appendix I. Similarly to the EMG training group, temperature trained subjects experienced a 5 minute adaptation period, a 2 minute baseline period, a 2 minute period with instructions to warm, all without feedback, followed by three 5 minute training sessions with feedback (Appendix J). If finger temperature was below 90°F, instructions during the training sessions were to continue warming until 90°F was

reached. If finger temperature was at or above 90°F at the beginning of the training session, instructions were to cool, and to move the response in the opposite direction in subsequent sessions. By this means, bi-directional control was taught to each subject after the acquisition of sufficient skill to warm digital temperature to 90°F at will.

For this group, attainment of criterion required 3 consecutive 5 minute periods of actually increasing and decreasing digital temperature by 2°F. Weaning procedures involved 3 practice sessions with feedback where temperature changes of 2 degrees were realized, followed by two sessions without feedback, when again temperature changes of a minimum of 2°F were realized. At this point subjects were considered to exhibit sufficient acquisition of the skill so that control could be exhibited at will without feedback, and training was considered to be completed. Subjects again received a minimum of four and a maximum of twelve sessions.

4.4.2 Follow-up Procedure

After approximately 4 weeks, a follow-up session was conducted with each subject. The format for the follow-up session was a repeat of the initial psychophysiological stress profile, and at this session, remaining daily headache monitoring charts were collected. The subject was thanked for participating in the study and encouraged to continue with newly learned relaxation skills. The experimenter indicated that a summary of the results of the experiment would be forwarded to each of the participating members at the conclusion of the project.

CHAPTER FIVE

5. RESULTS

5.1 Changes in headache intensity: Effects of Training Method and Group-reactivity

5.1.1 Reactivity and Treatment Groupings
5.1.2 Headache Intensity Variable
5.1.3 Analysis of Variance
5.1.4 Summary

5.2 Reduction in headache intensity from pre- to post-treatment in relation to training method and criterion-attainment

5.2.1 Criterion Measures -- EMG and Temperature
5.2.2 Statistical and Descriptive Analyses
5.2.3 Summary

CHAPTER FIVE

5. RESULTS

Two separate sets of data analysis are presented in order to consider the research questions under examination.

1. A three way analysis of variance with repeated measures on the last factor (C) was employed to analyze the headache activity ratings in the pre-treatment, treatment and post-treatment conditions for the two treatment methods (EMG treatment, Temperature treatment) and the two reactivity groups (EMG reactive, Temperature reactive).

2. The reduction in headache activity from period one (pre-treatment) to period three (post-treatment) was analyzed by a two (treatment method) by two (presence or absence of criterion attainment) factorial design to obtain the main effects of treatment method and criterion attainment, as well as their interaction.

5.1 Changes in Headache Activity; Effects of Training Method and Group Reactivity

5.5.1 Reactivity and Treatment Groupings

Reactivity groupings were based on the extent of EMG and temperature reactivity to stress as measured in the pre-treatment physiological stress profile (Carney, 1981). Reactivity was measured as a function of recovery from stress, recorded in seconds. Carney (1981) found that through the division of subjects into groups according to psychophysiological patterns of stress recovery, differential treatment effects were demonstrated. The rationale for such a division may be that physiological arousal during stress is adaptive, whereas staying aroused for a long period of time subsequent to the stressful event may be

maladaptive or unhealthy (Carney,]98]). EMG stress and recovery values as expressed in seconds were converted to z-scores and finally to T-scores. The dispersion of subjects according to these observations are presented in a scattergram (Figure 5-1). The diagonal line indicates the division of subjects into the two groups (a) those which are EMG reactive -- with relatively long EMG recovery periods and relatively short temperature recovery periods, and (b) those which are temperature reactive -- with relatively long temperature recovery periods and with relatively short EMG recovery periods. Each of these groups was further subdivided by a randomized block design procedure so that each group was divided into an EMG training group and a temperature training group. The results of Carney's study indicated that EMG treatment was more effective with temperature reactive subjects than temperature training, which, in turn, was shown to be more effective with EMG reactive subjects.

5.].2 Headache Intensity Variable

Daily headache ratings, as the dependent variable, were subsumed into three periods, P1 (pre-treatment period), P2 (treatment period) and P3 (post-treatment period). For each period, all headache ratings on a given day (Hd) were totalled (ΣHd), and divided by the number of days (D) in the period, so that (P), a mean headache rating/period was found. Therefore, $P=\Sigma Hd/D$, and each subject has a mean headache rating for P1, P2, and P3.

Headache data were subjected to two (groups) by two (treatments) by three (periods) repeated measures Analysis of Variance with subjects nested in treatments and groups. The research design is presented in Table 5-1.

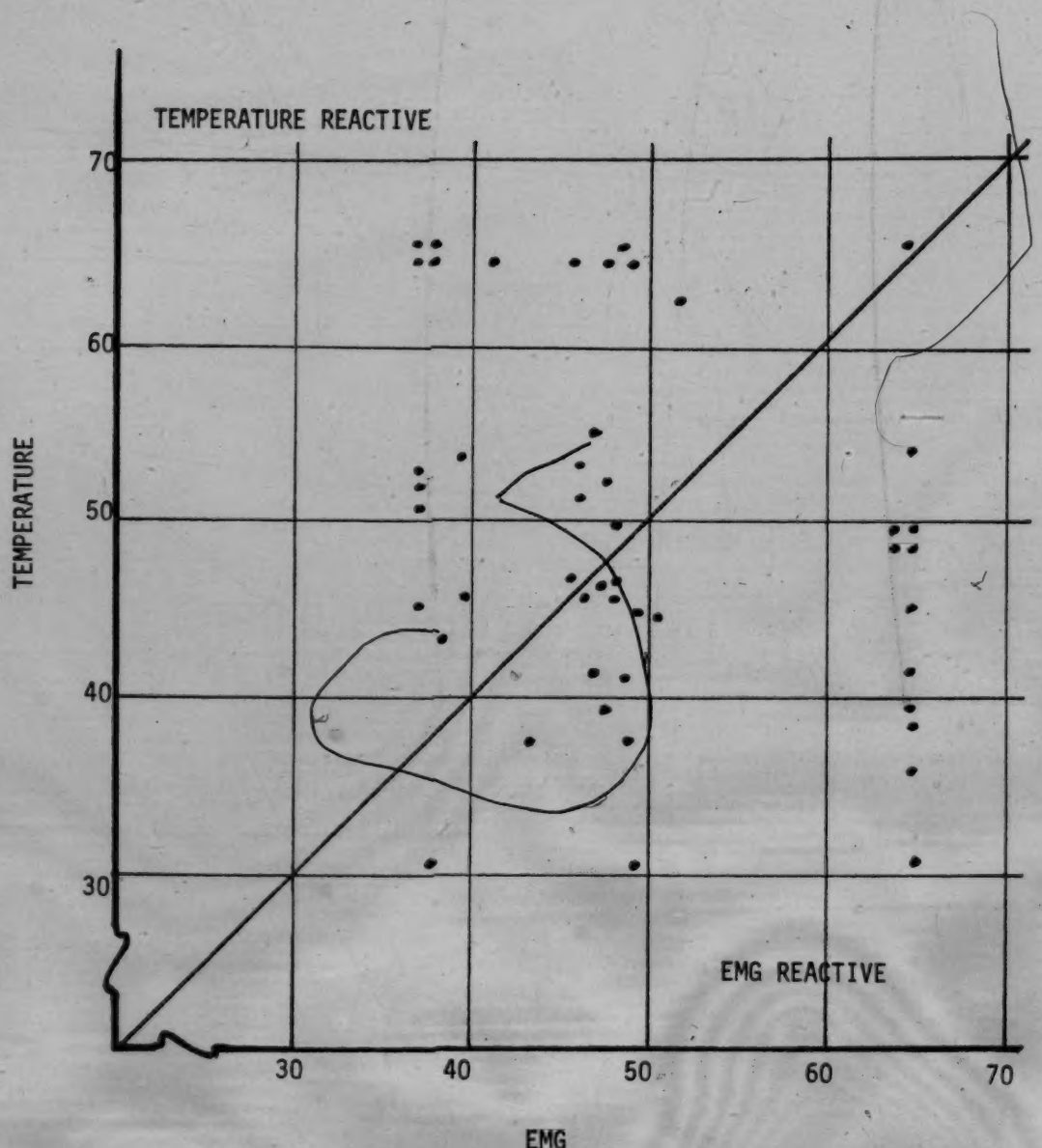


Figure 5-1

A Scattergram of EMG and Temperature T-Scores

Table 5-1

Distribution of Subjects by Groups and Treatment Methods (N=48)

		Experimental Period (C)		
Groups (B)	Treatment (A)	Pre-treatment P1	Treatment P2	Post-treatment P3
EMG Reactive	EMG Treatment	n=12	n=12	n=12
	Temperature Treatment	n=12	n=12	n=12
Temperature Reactive	EMG Treatment	n=12	n=12	n=12
	Temperature Treatment	n=12	n=12	n=12

5.1.3 Analysis of Variance

The Analysis of Variance indicated an overall significant period effect for the dependent measure, headache activity, $F(2,88)=9.6$, $p<0.001$. Results of the Analysis of Variance are outlined in Table 5-3. This significant period effect indicates that subjects obtained a significant reduction in migraine frequency and intensity regardless of the reactivity grouping or the type of biofeedback employed. No significant two-way or three-way interactions were obtained for the headache activity variables.

The Analysis of Variance indicated a significant difference in treatment effects, $F(1,44)$ 4.749, $p=0.035$, over the three periods, with EMG training exercising a greater influence on headache reduction than temperature training. No significant overall group effect was shown, which indicates that a difference in mode of stress reactivity did not affect responsivity to treatment. No significant interaction between treatment and group means was obtained. Treatment and group means are listed in Table 5-2 and presented graphically in Figure 5-2.

EMG training was associated with a headache reduction of approximately 50% for both EMG and temperature reactive groups. Temperature training resulted in a 40% reduction in headache activity for the temperature reactive group, and a 10% reduction in the EMG reactive subjects. It is noted that for the last group (temperature trained, EMG reactive), who experienced a smaller reduction in headache activity, headache activity means are considerably higher across all periods than those recorded in the remaining training groups.

Newman-Keuls post-hoc comparisons were made to determine the significant phases of headache reduction among the three treatment periods due

Table 5-2

Mean Headache Levels Across Reactivity Groups, Treatments and Periods

Reactivity	Treatment	Period P1	Period P2	Period P3
EMG Reactive	EMG	10.0	8.4	5.3
	Temp	17.9	19.1	16.2
Temp. Reactive	EMG	11.4	6.5	5.7
	Temp	10.7	9.9	6.4
Period Means (N=48)		12.5	11.0	8.4

Table 5-3

Summary of Analysis of Variance for Reactivity Grouping, Treatment Variable, and Headache Intensity Variable, Across Three Factor Repeated Measures (N=48)

Source	Sum of Squares	Degrees of Freedom	Mean Squares	F	P
A(Treatment)	1081.527	1	1081.527	4.749	0.035
B(Group)	691.573	1	691.573	3.037	0.088
AB(Interaction)	690.864	1	690.864	3.033	0.089
S-within	10020.910	44	227.748		
C(Period)	411.501	2	205.750	9.600	0.001
AC	69.905	2	34.953	1.631	0.202
BC	45.267	2	22.633	1.056	0.352
ABC	12.715	2	6.357	0.297	0.744
CS-within	1885.055	88	21.432		

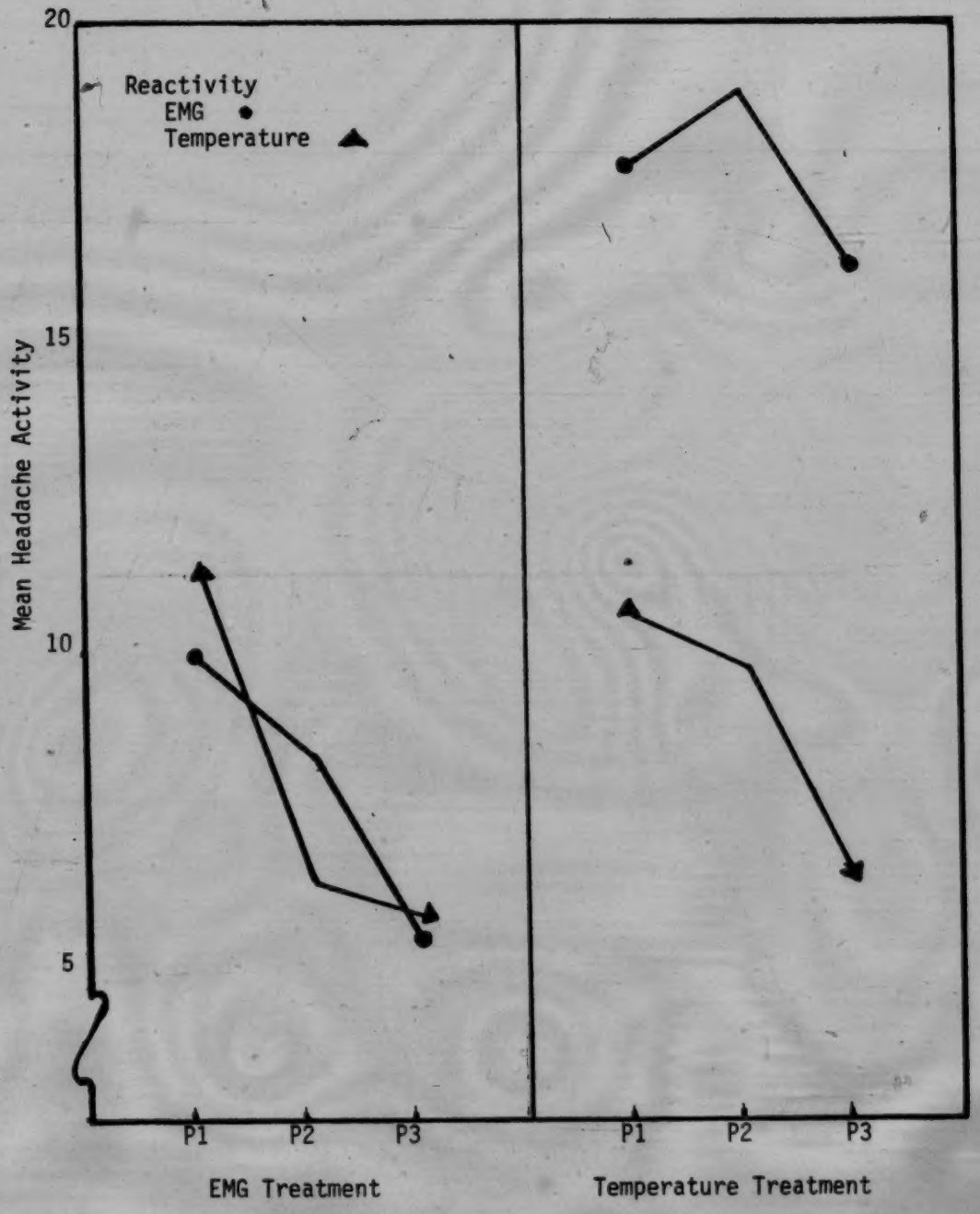


Figure 5-2

Mean Headache Activity Levels for the Two Treatments (EMG, Temperature) Two Groups (EMG Reactive, Temperature Reactive) and Three Periods (P1=pre-treatment, P2=treatment, P3=post-treatment)

to factor A (treatment effect) or factor B (group effect) on the means as shown in Table 5-4. When the group effect is collapsed, a significant reduction in headache was found from pre-treatment to post-treatment for the EMG trained subjects (C.diff.3=3.20). This group also showed a significant reduction between the pre-treatment and the treatment period (C.diff.2=2.66). Temperature trained subjects did not show a significant headache reduction from pre-treatment to post-treatment, but did demonstrate a significant headache reduction from the treatment period to post-treatment. When Neuman-Keuls comparisons were made between reactivity groups, not considering treatment methods, EMG reactive subjects demonstrated no significant headache reduction from pre-treatment to post-treatment (C.diff.3=3.20), but did demonstrate a significant change from treatment to post-treatment (C.diff.2=2.66). Temperature reactive subjects demonstrated a significant period effect from pre- to post-treatment, and demonstrated a significant change between pre-treatment and treatment periods, with no significant change demonstrated between treatment and post-treatment periods.

Summary

In summary, the Analysis of Variance indicates a significant reduction in headache activity over the three experimental periods with a significant treatment effect and no significant group effect. No significant interaction was found between any of the observed variables. EMG training methods were effective for both EMG and temperature reactive groups. While the temperature treatment was effective for the temperature reactive group, the EMG reactive group did not show a significant reduction in their migraine headache activity as a result of temperature training. This last group which

Table 5-4

Treatment and Period Means as Measured with Group Effect Collapsed and Group and Period Means As Measured with Treatment Effect Collapsed

AC		C= (Period)	1	2	3
			P1	P2	P3
A (Treatment)	1		10.686	7.463	5.497
	2		14.319	14.461	11.310
BC		C= (Period	1	2	3
			P1	P2	P3
B (Group)	1		13.936	13.735	10.771
	2		11.068	8.189	6.036

was more resistant to treatment effect, exhibited considerably higher headache activity across all treatment periods.

5.2 Reduction in Headache Activity from Pre- to Post-treatment Condition in Relation to Training Method and Criterion Attainment

5.2.1 Criterion Measures -- EMG and Temperature

Subjects who received EMG training received training on a bi-weekly basis until they were able to maintain relaxation levels of 1.5 microvolts (or less) for three consecutive 5 minute training periods. Twenty-two of the twenty-four subjects who received EMG training met this criterion within the allowed period of twelve training sessions.

Subjects who received digital skin temperature training were trained to a criterion which required the demonstration of finger temperature control of an upward or downward change in temperature of 2°Fahrenheit over a period of three consecutive training periods, each of 5 minute duration. Eleven of the twenty-four subjects who received temperature training met this criterion within the alloted period of twelve training sessions.

5.2.2 Statistical and Descriptive Analysis

The Analysis of Variance indicated no significant relationship between reduction in headache activity and the attainment of criterion for EMG or temperature training methods (Table 5-5). Further to the Analysis of Variance, Pearson Product Correlation coefficients were calculated, based on comparisons of mean EMG level reductions from pre- to post-treatment periods and pre- and post-headache activity means (Table 5-6). EMG relaxation level changes from pre- to post-treatment were calculated by taking the means of the EMG readings taken at 20 second intervals during the last 5 minutes of the 15 minute relaxation

Table 5-5

Summary of Analysis of Variance for Treatment Variable, Attainment (non-attainment) of Criterion, Headache Intensity Variables, Two-Factor ANOVA (N=48)

Source of Variation	Sum of Squares	DF	Mean Square	F	Significance of F
Main Effects	98.202	2	49.101	0.922	0.405
TR	11.511	1	11.511	0.216	0.644
CRIT	41.162	1	41.162	0.773	0.384
2-WAY INTERACTIONS	61.947	1	61.947	1.163	0.287
TR CRIT	61.947	1	61.947	1.163	0.287
EXPLAINED	160.148	3	53.383	1.002	0.401
		44	53.257		
RESIDUAL	2343.302	47	53.265		
TOTAL	2503.450				

Table 5-6

Pearson Product Correlation Coefficients Based on a Comparison of the Difference Between Pre- and Post-Baseline EMG (E1-E3) Measurements and Pre- and Post- Baseline Temperature (T2-T1) Measurements and Headache Activity Reduction (P1-P3)

	BT	BE	013
BT	1.000 (24) P=****	0.0124 (24) P=0.477	-0.3974 (24) P=0.027
BE	0.0124 (24) P=0.477	1.0000 (24) P=****	0.0961 (24) P=0.328
013	-0.3974 (24) P=0.027	0.0961 (24) P=0.328	1.0000 (24) P=****

time in the pre- and post-psychophysiological stress profiles. The difference (E1-E3), when correlated with the difference between Period 1 and Period 3 (P1-P3) demonstrated a correlation of r=0.1651. In the same manner, temperature readings were averaged in the last 5 minutes of the 15 minute relaxation time in Period 1 (pre-treatment) and Period 3 (post-treatment). The mean (T1) was subtracted from the mean (T2), as it was theorized that relaxation training would increase the peripheral temperature during relaxation. In this correlation, r=-0.1804, a negative correlation which indicates that the post-treatment baseline relaxation temperature was not higher but lower than the pre-treatment baseline relaxation temperature, contrary to expectations. In the correlation of the mean (E1-E3) and the mean (T2-T1), r=-0.0827. These results indicate very little correlation between headache reduction and physiological measures of relaxation, nor is any notable correlation between the two physiological systems evidenced.

It is noted, however, that in the statistical correlations just described, reductions in headache activity and changes in EMG and temperature are measured in period changes from pre- to post-treatment. When the data is examined descriptively and headache activity and EMG and temperature baseline changes are measured in a percentage of change, some interesting effects emerge. A comparison of mean changes in headache activity indicate that for EMG trained subjects, the mean headache reduction was greater for those subjects who did not reach criterion (Figure 5-3). However, further examination of the data reveals that 92% of EMG trained subjects reached criterion and that this group experienced an average headache reduction of 50%. Since the remaining 8% who did not reach criterion and who experienced a 74% reduction in headache

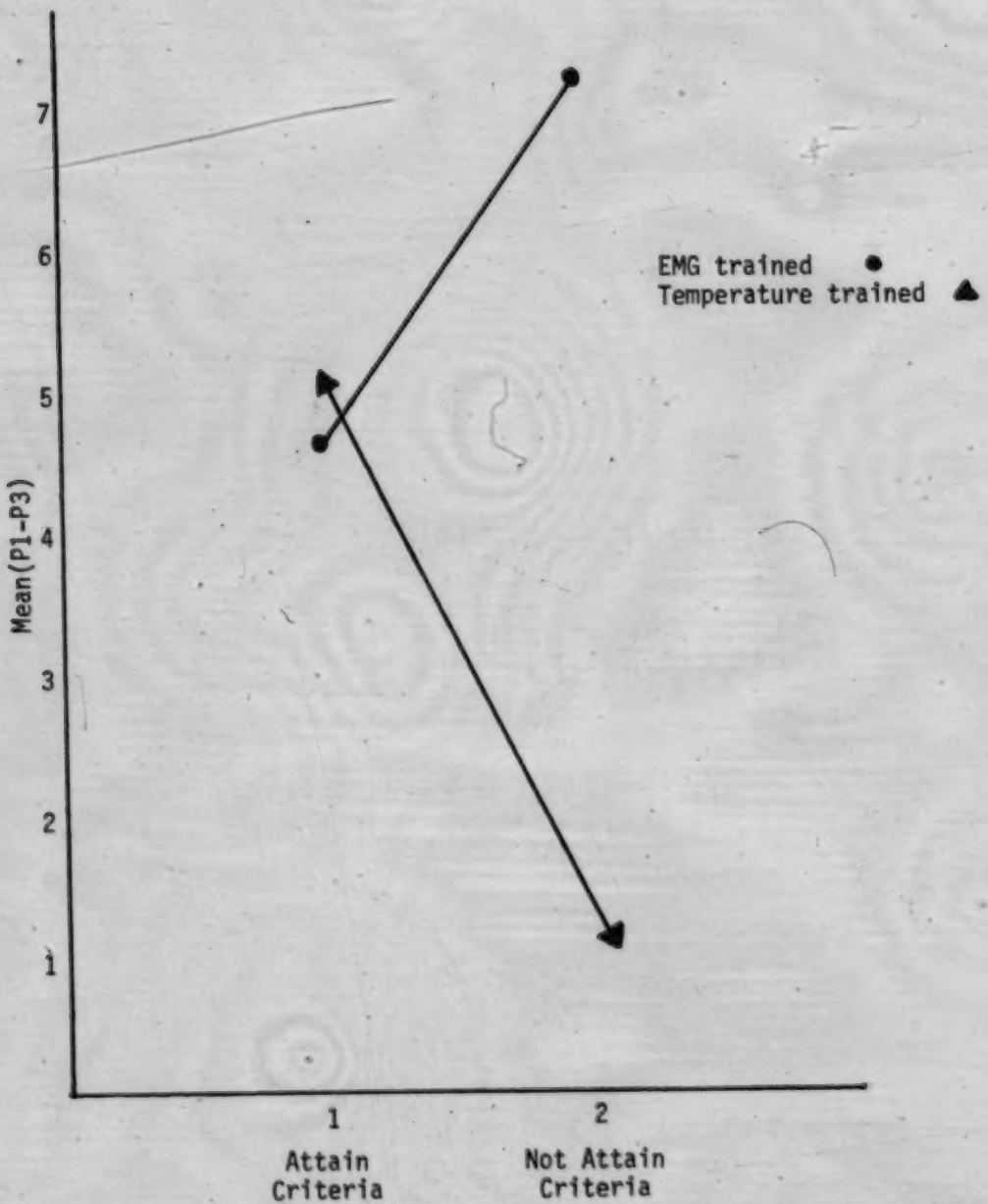


Figure 5-3

Mean Headache Activity Reductions (P1-P3) for the Two Treatments (EMG, Temperature) for Subjects who Attained Criterion and for Subjects who did not Attain Criterion

activity represent only two subjects, comparison are relatively meaningless. The general 50% headache reduction for EMG trained subjects is accompanied by a 47% reduction in EMG levels from pre-treatment baseline relaxation levels (M=2.05Mv), to post-treatment baseline relaxation levels (M=1.08Mv), as shown in Table 5-7. These same subjects demonstrated a slight reduction in baseline skin temperature from Period 1 to Period 3 (pre- to post-treatment).

A further examination of Figure 5-3 indicates that for temperature trained subjects, those who attained criterion demonstrated a mean headache reduction from pre- to post-treatment of 5.10, and those who did not, experienced a mean headache reduction from pre- to post-treatment of 1.24. Among the temperature trained subjects, 46% (eleven subjects) attained criterion and experienced a 26% reduction in headache activity from pre- to post-treatment. The remaining 54% of temperature trained subjects (thirteen subjects) who did not attain criterion experienced a 14% reduction in headache activity from pre- to post-treatment. Temperature trained subjects demonstrated a mean EMG baseline reduction from pre- to post-treatment of 0.62Mv (Table 8), a figure which represents a 27% reduction. Temperature trained subjects also exhibited a slight drop in pre- and post-treatment baseline temperatures.

For all subjects, the mean baseline EMG level fell from 2.18 Mv in pre-treatment to 1.38 in post-treatment, a 37% reduction which suggests that relaxation training was effective overall in reducing EMG levels during periods of relaxation. An examination of pre- and post-baseline temperature levels during relaxation for all subjects reveals a drop from a mean of 92.6° Fahrenheit to a mean of 90.2° Fahrenheit (Table 5-6). Relaxation training did not, then, result in increased peripheral skin temperature under conditions of relaxation, as expected. Thus, a

Table 5-7

Mean EMG and Temperature Baseline Levels as Measured During Final 5 Minutes of Relaxation Prior to Stressing in Pre (P1) and Post (P3) Treatment Periods

		EMG Trained	Temperature Trained	Total
Mean EMG Levels in Mu	P1	2.05	2.30	2.18
	P3	1.08	1.68	1.38
Difference		0.97	0.62	0.80

		EMG Trained	Temperature Trained	Total
Mean Temperature Levels in Degrees Fahrenheit	P1	93.2	92.1	92.6
	P2	89.5	90.9	90.2
Difference		3.7	1.2	2.4

negative correlation in temperature change and headache activity is seen.

5.2.3 Summary

The Analysis of Variance did not demonstrate any overall significant effect when subjects were compared who mastered the attainment of a preset experimental criterion, as against those who did not. Pearson Product-moment Correlations show little correlation between the difference in pre- and post-headache activity and the difference in pre- and post-EMG or temperature baseline relaxation levels. Correlations between baseline levels of the two physiological measures, EMG and peripheral skin temperature, are negligible. A descriptive examination of the data indicates that EMG trained subjects were able to master the preset criterion more readily than those who were temperature trained. Within training groups, EMG trained subjects who attained criterion achieved a lower headache reduction than those who did not. This comparison is based on uneven numbers in groups, as only two subjects, who were both relatively successful in achieving headache reduction, failed to reach criterion. Temperature trained subjects who attained criterion achieved greater levels of headache reduction than those who did not, which suggests that criterion attainment may be of some importance in temperature training. Training effects are associated with lowered levels of basal EMG readings, and slightly lowered levels of basal skin temperature.

CHAPTER SIX

6. DISCUSSION

6.1 Overview

6.2 The Research Questions

6.2.1 Question One

6.2.1.1 Summary

6.2.2 Question Two

6.2.2.1 Summary

6.2.3 Question Three

6.2.3.1 Summary

6.3 Practical and Theoretical Implications

6.4 Related Research

CHAPTER SIX

6. DISCUSSION

6.1 Overview

This chapter discusses the major findings of the study relevant to the research questions directly under consideration. Following the examination of results as related to research questions one and two, the hypothesized influence of psychological mechanisms are discussed in response to question three. The results are then discussed in relation to some of the practical and theoretical implications of biofeedback training in migraine headache. This discussion is concluded with an examination of the implications for further research.

6.2 The Research Questions

The design of this investigation was developed to consider three specific questions. Each of these will now be addressed.

6.2.1 Question One

What are the effects of skin temperature training versus frontalis electromyographic control on headache activity?

Biofeedback training, as employed in this study, was effective in reducing headache activity among the experimental subjects. The Analysis of Variance demonstrated a differential treatment effect where frontalis EMG training was seen to be more effective than skin temperature training. For the two EMG trained groups, treatment effects were similar and generally effective. Temperature training was generally less effective, and was particularly ineffective for the EMG reactive, temperature trained group. Observation of mean headache activity levels for the two treatment groups indicates that

for all EMG trained subjects, and for the temperature reactive, temperature trained group, pre-treatment headache activity means were comparable in range. Mean pre-treatment headache activity levels for the EMG reactive, temperature trained group were considerably higher than the means for any other of the treatment groups. This differential effect may have occurred as a result of sampling, or may be an indication that temperature training is generally less effective for subjects who demonstrate a relatively high mean headache activity. It is hypothesized that these relatively high headache activity levels may occur through muscular tension rather than vascular mechanisms and, therefore, be less responsive to temperature training methods. Particular psychological and social factors may be involved in the maintenance of such prolonged and intense headache. Whether through psychological change or physiological control, such subjects may only demonstrate substantial treatment effects through more intensive biofeedback training, with the inclusion of EMG relaxation training and formal psychotherapy. De Good et al. (1978) present evidence that success in application of skills learned in EMG biofeedback sessions is related to the number of training sessions included in the program, and researchers such as Mitchell and Mitchell (1971) and Paulley and Haskell (1975) strongly support the inclusion of psychotherapy in migraine headache treatment programs.

Biofeedback techniques, then, are shown to be effective but their mode of action is uncertain. A discussion of previous studies, as presented earlier in this paper, has examined treatment outcomes of biofeedback training in terms of specific or general relaxation effects. Based on previously examined theoretical considerations and

the treatment results in the present study, some hypotheses regarding the mode of action of biofeedback training may be presented.

In the application of biofeedback, migraine sufferers have traditionally been treated through temperature training procedures (Sargent, Green, & Walters, 1972) and frontalis EMG training has been administered to tension headache sufferers (Budzynski, Stoyva, & Adler, 1970). But the work of Bakal and Kaganov (1977) suggests that there is no sharp delineation in diagnosis between migraine and tension headache. Their study found that individuals who were diagnosed to suffer from tension headache experienced a number of symptoms characteristic of migraine, and that migraine patients also have higher EMG values both during headache and during headache-free intervals than muscle contraction patients or controls. Bakal and Kaganov (1977) further observed that migraine and muscle contraction patients have higher neck EMG activity than controls. Such observations led Bakal and Kaganov to conclude that there is no specific type of headache of muscular origin and that sustained muscle activity may be predisposing for both migraine and muscle contraction headaches. Such conclusions suggest the application of muscle relaxation training in any functional headache.

Kappes and Morris (1981) conducted a study which applied EMG biofeedback, temperature biofeedback and relaxation training to adult volunteers and measured physiological changes subsequent to training. Temperature and EMG results were found to be not significantly related. Such results infer independence of the two physiological systems. The work of Kappes and Morris supports the results of the present study which finds no significant correlation between alteration of

peripheral blood flow and voluntary muscular relaxation.

Arnarson and Sheffield (1980), in a study which applied EMG and temperature biofeedback to patients with high anxiety, found frontalis EMG to reduce activity in a group of muscles related to the frontalis and found this effect to generalize in reducing sympathetic activity. In this study temperature biofeedback was seen to be less efficient, but better than a control procedure. This study supports the generalization effect of EMG training, and the efficiency of EMG training as compared to temperature training.

Clinical treatment programs in stress management often provide a combination of biofeedback training modalities and concomitant relaxation training. The use of a combination of biofeedback techniques may be warranted on the evidence that through individual differences in certain cases one method may be successful where another is not, as evidenced in the work of Wickrameskera (1973). Where such procedures are followed, the suggested sequence is EMG training followed by temperature training. Kappes and Morris (1981) reported that those groups which received temperature training prior to EMG training reported dissatisfaction initially because success was difficult, and results of the present study suggest that temperature control is more difficult to master, and less effective overall than EMG training in headache control.

On the basis of psychophysiological stress data which separated subjects into EMG and temperature stress reactive groupings, no significant differences in training effect on headache activity was shown. The results of this study do not support those of Carney (1981) who reported differential effectiveness in training among the two stress

reactivity groups. He reported that frontal EMG biofeedback was more effective with subjects who demonstrated relatively slow temperature recovery from stress, and relatively fast EMG recovery from stress. Carney reported that temperature biofeedback was more effective in reducing headaches with subjects who demonstrated relatively slow EMG recovery and relatively fast temperature recovery. On the basis of physiological stress recovery differences, this present study does not favor the use of a particular training modality.

6.2.1.1 Summary

In summary, it is concluded that muscular tension plays an important (predisposing) role in migraine as well as in tension headache. Such 'bracing' effects are seen as a form of dysponesis and as a function of overstimulation of the sympathetic nervous system. Sympathetic activity can be reduced through the learning and application of systematic muscular relaxation procedures. Since biofeedback has been demonstrated to enhance the learning of muscular relaxation (Reinking & Kohl, 1973), it is concluded that EMG training is an effective mode of treatment for migraine headache. It is further proposed that training in the control of peripheral blood flow is less effective as a treatment modality, as the smooth musculature (as is found in the blood vessels) is controlled through parasympathetic innervation, a physiological system which plays a lesser role in the dysponetic state. It is suggested that peripheral temperature control may be effective for the proportion of the population who are able to learn this skill, but that this effect occurs primarily through a hydraulic shift in blood flow, or through the existence of separate

pain mechanisms as some researchers suggest (Sturgis, Tollison & Adams, 1978; Build & Adams, 1980). Temperature control is therefore seen to exert its effect primarily on a specific aspect of the migraine. If the migraine itself is viewed as secondary to the general state of dysponesis, such treatment will be, by nature, less efficacious than an attack on the primary source of the dysfunction. The present study does not support the theory that differences in physiological stress recovery mechanisms affect responsivity to biofeedback treatment.

6.2.2 Question Two

What is the relationship between the attainment of a predetermined criterion and headache reduction?

The Analysis of Variance indicated no significant relationship between reduction in headache activity and the attainment of criterion for EMG or temperature training methods. No empirical evidence, then is provided to support the process of training to a pre-set experimental criterion. Examination of the data indicates that the pre-set experimental criterion was attained by a higher percentage of EMG trained subjects as compared to those who were temperature trained. Twenty-two of twenty-four EMG trained subjects reached criterion as compared to eleven of twenty-four temperature trained subjects. Only two of the EMG trained subjects failed to reach criterion and these two subjects were particularly successful in achieving a reduction in headache activity. Since only two subjects were included in one of the cells under analysis, the validity of the Analysis of Variance is under question, and statistical relationships are confounded.

In the absence of empirical evidence that EMG training to criterion

is important in achieving headache reduction, it may be argued that such a procedure is antithetic to successful treatment. The subjects who achieved criterion received fewer training sessions than those who did not. As previously reported, DeGood et al. (1978) conducted a study which manipulated "length of training" periods for a group of subjects with functional disorders and reported that while EMG levels were not greatly reduced by increasing the number of training sessions, therapeutic effects were stronger for those subjects who received more training sessions. DeGood et al. (1978) conclude that additional training may be necessary for the subject to consolidate the internal response which permits the transfer of relaxation skills to outside situations. These results support the consensus that biofeedback is not a 'quick relief' program, but that improvement in physiological self-regulation is a gradually acquired skill.

In addition to the above considerations, subjects were aware that the training process was geared to the attainment of criterion and thus became involved in 'trying hard' to attain this goal. Such a response tended to interfere with the process of 'passive concentration', a process which is an essential requisite to the achievement of lower EMG readings and true relaxation. Furthermore, in the absence of evidence to support a direct linear correlation between measured EMG readings and headache reduction, the rationale behind EMG training to a pre-set experimental criterion is under question.

In the case of temperature training, examination of mean headache activity during the experimental period (Figure 5-2) and the results of the Newman-Keuls post-hoc tests show that temperature trained subjects experienced considerable headache reduction between the treatment

period and the post-treatment period, although overall treatment effects were not significant. This treatment effect may indicate that for these subjects, such headache reductions as were realized occurred upon the attainment of criterion. One may hypothesize from these results that through temperature training, a subject may be primarily learning to control peripheral blood flow, reducing intra and extracranial pressure on dilated arteries, and thus alleviating the symptoms of his/her headache as suggested in the discussion of Question One. Therefore, the attainment of the experimental criterion, which indicates a learned control of blood flow, may be of some importance in the temperature training procedure. It is reiterated that direct symptom control, overall, is considered to be a less effective process than the enhancement of a general relaxation response.

6.2.2.1 Summary

In summary, there is no empirical evidence to support the procedure of training to criterion in the modalities of EMG or temperature biofeedback training. It is theorized that EMG training should not be directed to a pre-set criterion and that EMG training may be, in fact, more effective when increased numbers of training sessions are included in the program. It is further hypothesized that for temperature trained subjects, the attainment of criterion, which indicates learned temperature control and hence the control of peripheral blood flow, may be a desirable goal when biofeedback assisted temperature training is applied for the purpose of migraine headache relief.

6.2.3 Question Three

What non-specified variables in the study may influence subject

headache reduction?

In Chapter Three, it was suggested that certain psychological variables may affect biofeedback learning and associated reductions in pain. Such variables, commonly categorized as placebo effects, are seen to include subject attitudes, motivation, and experimenter influences. An examination of these variables is presented in an anecdotal manner, as these factors were not controlled for in the present study.

In general, subjects initially volunteered for the biofeedback program with only a limited awareness of what the program involved. Most subjects have suffered from debilitating migraine attacks for a number of years, had explored other methods of treatment, and had experienced only limited relief through drug therapy. A number of these subjects expressed concern regarding the effects of continued drug use on their general health. The fact that the present program was offered as part of a research project, using sophisticated instrumentation, at the University of Alberta appeared to lend some credibility to the procedures. Upon learning that biofeedback involved a rather simplistic training method, rather than an applied 'treatment', the success of which depended on their own efforts, a number of the subjects expressed skepticism, but felt that for the small fee involved, it was worth a 'try'.

Initial doubt, however, frequently led to positive results. As many migraineurs tend to be self-disciplined, organized and perfectionistic, headache records were meticulously maintained and relaxation exercises were practised scrupulously in most cases, in spite of any skepticism which may have existed regarding the procedures. A number

of subjects, in fact, discovered that merely through keeping the headache records, they became more aware of the frequency and the causes and effects of their migraine attacks. One subject recognized through such record keeping that his headaches were associated with the ingestion of fatty foods. Upon the elimination of such foods from his diet, his headache activity was reduced -- and he dropped out of the program.

The skeptical, also, were less inclined to 'try too hard' in the actual training period. As has been previously stated, success in the use of biofeedback requires a 'passive volition' (Sargent, Green & Walters, 1972), an attention without effort. When such a passive attitude is achieved, control of frontalis muscular activity occurs relatively easily and rapidly, while temperature control is gained more slowly and unevenly. Those subjects, then, many of whom were initially skeptical, who did achieve success in the training sessions, and who practiced relaxation techniques regularly, expressed excitement in the discovery of personal control, and joy in the achievement of positive changes in lifestyle with concurrent headache reduction. It is noted that such effects were gained, in many cases, through biofeedback and relaxation training only, in a maximum of twelve bi-weekly training sessions. No formal psychotherapy was included in the program, although experimenters applied such counselling techniques as were judged to be appropriate. Subject and experimenter interactions, therefore, are considered to have facilitated the training process.

Such success was not universal, however. A lack of faith may lead a subject to give up. One female subject of fifty-nine years, who was experiencing little success, expressed continuing doubts regarding

her own ability to achieve the control that she judged others to be gaining easily. When the experimenter dropped her normally supportive and encouraging attitude and expressed to this subject firmly that success could only be achieved through a belief in her own ability and through a positive attitude, this subject, who was receiving temperature training, achieved criterion within three more training sessions.

A certain percentage of subjects, some of whom had high expectations of themselves and the program or who were intent on following directions, were unable to achieve the relaxed concentration necessary for success. When a subject tried too hard, or 'braced' with effort, results were counter-productive. EMG levels remained high, and temperature levels resisted control and plumetted lower and lower. In another instance, a subject who had achieved a high level of proficiency in voluntary peripheral temperature control, was able to exhibit no control whatever immediately following a prolonged and severe headache. It is hypothesized that neural and biochemical factors mitigated against voluntary control in this instance.

Psychological and social factors were observed to play a role in the maintenance of headache for some subjects. For one female who suffered from severe and continuous head pain, the 'headache' appeared to be a 'focal point' in her life. She gained attention from family members, friends, physicians, and biofeedback clinicians, as a result of her head pain. Difficult life situations could be avoided, through constant 'illness'. Biofeedback training was not effective for this subject.

However, environmental pressures may genuinely affect success in biofeedback training. A young female subject practised relaxation

exercises faithfully and attained impressive peripheral temperature control with no accompanying headache reduction. This young woman was raising a young child alone, under difficult financial and social conditions. It is judged that while an enhanced ability to relax was beneficial to this subject, further life changes would be necessary in order to reduce her somatic symptoms.

Biofeedback is seen as a superb means by which a subject is introduced to the concept that he/she is responsible for his/her own symptoms. Initially, the subject is not defensive in approaching biofeedback training, as an applied 'cure' is expected. As the process of biofeedback assisted learning and relaxation proceeds, subjects generally appear to gradually accept a responsibility for their own symptomology, and to relate this symptomology to lifestyle -- a lifestyle which only they can change. In this process, subjects were observed to respond well to reinforcement, encouragement and support provided by the experimenters. Thus, biofeedback training appears to facilitate a general shift in attitude from 'helplessness' to 'resourcefulness'.

Age was not seen as a factor in biofeedback performance, as training did not become less effective with older subjects. Subjects above the median age demonstrated a 48% reduction in headache activity, and those below demonstrated a 43% reduction in headache activity. The median age of thirty-eight indicates that most subjects who participated in this study were within the middle age bracket.

One other unspecified factor deserves mention. In this study, subjects were trained in pairs. The question is raised as to whether the presence or performance of one subjects affects the other. Hendricks

(1977) describes an experiment performed by Charles Tart of the University of California in which a subject was connected to a plethysmograph (to monitor blood flow), an electroencephalograph, and a device that records galvanic skin response. A second subject was shocked at random intervals, and the first subject was asked if he could tell when the shocks were administered. His responses were completely at random, but his physiological measurements did respond whenever the second subject was shocked. Such results indicate that communication works body to body, and that such influences may have exerted some effect on the performance of subjects in our study.

6.2.3.1 Summary

In summary, a discussion of non-specific variables in biofeedback training has included psychological and social considerations. Psychological factors considered have included subject attitudes toward treatment, and their motivations to succeed. Situational life conditions are also considered as factors which may influence biofeedback performance and associated changes in headache activity. Interaction between subject and clinician and between subject and subject is also considered as a possible factor in the experimental process. The above mentioned variables are seen to influence biofeedback performance to an unspecified degree.

6.3 Theoretical and Practical Implications

The present study verifies the general effectiveness of biofeedback training, as has been demonstrated in previous experimental studies. To date, conflicting evidence has been assembled as to the relative superiority of EMG training, as opposed to temperature training. The results

of this study indicate that frontalis EMG training is superior to peripheral skin temperature training in the treatment of migraine, and a theoretical rationale is presented to account for these results, although higher headache activity means may have tempered the beneficial effects of temperature training. The practical implications of the results obtained should therefore be considered to be limited until replicated by future studies. Additional factors which may limit the import of the present study include the size of the sample used, the number of treatment sessions provided, and the limitation of the follow-up period to one month. It is suggested that with a larger sample size, with a more prolonged treatment and follow-up period, results of greater significance would be obtained. Meanwhile, common clinical practice which includes two biofeedback training methods in the treatment of migraine headache would seem to be supported. This study provides no empirical evidence that any one form of training is superior to another on the basis of the individual physiological difference. Through anecdotal evidence, it is implied that psychological differences may influence the efficacy of biofeedback training.

With regard to theoretical implications, the results and conclusions of this study support the theoretical position of Whatmore and Kohli (1974) who view increased muscular tension as the primary manifestation of overactive sympathetic arousal, a process which leads to altered circuit activity within the nervous and neuromuscular systems and consequent alterations in tissue and organ function. This view leads to the use of electromyography and muscular relaxation training as the treatment of choice in alleviating the development of neuromuscular physiopathology and subsequent functional disorders.

6.4 Related Research

Further research is necessary to verify the results and conclusions of the present study. It is suggested that future research designs be based on subject groupings with comparable headache activity levels, and that pre-treatment interviews be held in an attempt to isolate those psychological factors which may influence the efficacy of bio-feedback training. It is further suggested that the power of such studies be increased with larger sample sizes, and that in EMG treatment the number of training sessions be related to the criterion of symptom alleviation rather than a specific microvolt reading. It is also proposed that the effects of experimenter and subject interaction, and/or the inclusion of formal psychotherapy as an adjunct treatment be measured. The value of any treatment program lies truly only in the attainment of long-term effects, therefore it is further suggested that research studies include follow-up periods of greater duration.

REFERENCES

Ad Hoc Committee on Classification of Headache. Classification of headache. American Medical Association Journal, 1962, 179, 717-718.

Adams, H.E., Feurstein, M., & Fowler, J.L. Migraine headache: Review of parameters, etiology, and intervention. Psychological Bulletin, 1980, 87, 217-237.

Alvarez, W.C. The migrainous personality and constitution. American Journal of Medical Sciences, 1947, 213, 1-8.

Andreychuk, T., & Skriver, C. Hypnosis and biofeedback in the treatment of Migraine headaches. The International Journal of Clinical and Experimental Hypnosis. 1975, 23(3), 172-183.

Anthony, M., & Lance, J.W. Current concepts in the pathogenesis and interval treatment of migraine. Drugs, 1972, 3, 153-158.

Anthony, M. Plasma free fatty acids and prostaglandin E, in Migraine and stress. Headache, 1975, 16, 58-63.

Anthony, M., Hinterberger, H., & Lance, J.W. Plasma serotonin in migraine and stress. Archives of Neurology, 1967, 16, 544-554.

Anthony, M., & Lance, J.W. The role of serotonin in migraine. In Modern Topics in Migraine, J. Pearce (Ed.), London: William Heinemann Medical Books Ltd., 1975.

Arnarson, E.D. & Sheffield. The generalization of the effects of EMG and temperature biofeedback procedures in patients suffering from anxiety states. Proceedings of the Biofeedback Society of America, Eleventh Annual Meeting, March, 1980, 5-6.

Bakal, D.A. Headache: A biopsychological perspective. Psychological Bulletin, 1975, 82, 369-382.

Bakal, D.A. & Kaganov, J.A. Muscle contraction and migraine headache: Psychophysiologic comparison. Headache, 1977, 17, 208-214.

Basmajian, J.V. Principles and Practice for Clinicians. Baltimore: Williams & Wilkins, 1979.

Benson, H. The Relaxation Response. New York: Hearst, 1975.

Bild, R. & Adams, H.E. Modification of migraine headaches by cephalic blood volume pulse and EMG biofeedback. Journal of Consulting and Clinical Psychology, 1980, 48(1), 51-57.

Birk, L. Biofeedback: Behavioral Medicine. New York: Grune & Stratton, 1973.

Blanchard, E.B., Theobald, D.E., Williamson, D.A., Silver, B.V. & Brown,

D.A. Temperature biofeedback in the treatment of migraine headaches. Archives of General Psychiatry, 1978, 35, 581-588.

Budzynski, T.H., Stoyva, J.M., Adler, C.A. & Mullaney, D.J. EMG biofeedback and tension headache: A controlled outcome study. Psychosomatic Medicine, 1973, 35, 484-496.

Budyznsky, T.H., Stoyva, J.M., & Adler, C.S. Feedback-induced muscle relaxation: Application to tension headache. Behavior Therapy and Experimental Psychiatry, 1970, 1, 205-211.

Carlson, J.G. Locus of control and frontal electromyographic response training. Biofeedback and Self Regulation, 1977, 2(3), 259-271.

Carney, P. Psychophysiological Stress, Biofeedback and Migraine Therapy. Unpublished thesis, University of Alberta, 1981.

Chapman, L., Ramos, A.O., Goodell, H., Silverman, HG., & Wolff, H.G. A humoral agent implicated in vascular headache of the migraine type. Archives of Neurology, 1960, 3, 223-229.

Chesney, M.A. & Shelton, J.L. A comparison of muscle relaxation and electromyogram biofeedback treatments for muscle contraction headaches. Journal of Behavior Therapy and Experimental Psychiatry, 1976, 7, 221-225.

Cox, D.J., Freundlich, A., & Meyer, R.G. Differential effectiveness of electromyograph feedback, verbal relaxation instructions, and medication placebo with tension headaches. Journal of Consulting and Clinical Psychology, 1975, 43(6), 892-898.

Cummings, C. & Trabin, T.E. Locus of control and patient's attitudes towards biofeedback, relaxation training and group therapy. American Journal of Clinical Biofeedback, 3(2), 144-147.

Daniels, L.K. The effects of automated hypnosis and hand warming on migraine: A pilot study. The American Journal of Clinical Hypnosis, 1976, 19(2), 91-94.

DeGood, D., Tennenbaum, D. & Greenwald, D. "Length of Training" as a factor in the Biofeedback Treatment of Psychosomatic Symptoms. Proceedings of the Biofeedback Society of America, 9th Annual Meeting, 1978, 88-91.

Diamond, S. & Dalessio, D.J. The Practicing Physician's Approach to Headache, Baltimore: Williams & Wilkins, 1978.

Dukes, H.T. & Vieth, R.G. Cerebral arteriography during migraine prodrome and headache. Neurology, 1964, 14, 636-639.

Fanchamps, A. The role of humoral mediators in migraine headaches. Canadian Journal of Neurological Sciences, 1974, 1, 189-195.

Friar, L.R. & Beatty, J. Migraine: Management by trained control of vasoconstriction. Journal of Consulting & Clinical Psychology, 1976, 44(1), 46-53.

Friedman, A.P. Migraine. Psychiatric Annals, 1975, 5, 271-278.

Friedman, A.P. Prophylaxis of migraine. Headache, 1973, 13, 104-106.

Friedman, A.P., von Storch, T.J.C., & Merritt, H.H. Migraine and tension headaches - A clinical study of two thousand cases. Neurology, 1954, 4, 773-788.

Fromm-Reichmann, F. Contributions to the psychogenesis of migraine. Psychoanalytic Review, 1937, 24, 26-33.

Furmanski, A.R. Dynamic concepts of migraine. Archives of Neurology and Psychiatry, 1952, 67, 23-31.

Gray, H. Anatomy, descriptive and surgical. Philadelphia: Running Press, 1975.

Green, E.E., Walters, E.D., Green, A.M. & Murphy, G. Feedback techniques for deep relaxation. Psychophysiology, 1969, 6, 371-377 (R:48)

Harris, F.A. Multiple loop modulation of motor outflow: A physiological basis for facilitation techniques. Journal of the American Physical Therapy Association, 1971, 51, 391-397.

Hendricks, G. & Roberts, T. The Second Centering Book. Englewood Cliffs: Prentice-Hall, 1977.

Henryk-Gutt, R. & Rees, W.L. Psychological aspects of migraine. Journal of Psychosomatic Research, 1973, 17, 141-153.

Hicks, R.G. Experimenter effects on the physiological experiment. Psychophysiology, 1970, 7, 10-17.

Horrobin, D.F. Hypothesis: Prostaglandins and migraine. Headache, 1977, 17, 113-117.

Jacobson, E. Electrical measurements concerning muscular contraction (tonus) and the cultivation of relaxation in man: Studies on arm flexors. American Journal of Physiology, 1933, 107, 230-248.

Jencks, B. Your Body, Biofeedback at Its Best. Chicago: Nelson Hall, 1977.

Johnson, W.G. & Turin, A. Biofeedback treatment of migraine hadache: A systematic case study. Behavior Therapy, 1975, 6, 394-397.

Kappes, B.M. & Morris, R.C. The sequence of effects of relaxation training, EMG, and temperature biofeedback on anxiety, symptom report and self-concept. Proceedings of the Biofeedback Society

of America, Twlefth Annual Meeting, March, 1981, 50-51.

Keefe, F.J. Conditioning changes in differential skin temperature. Perceptual and Motor Skills, 1975, 40, 283-288.

Kolb, L.C. Psychiatric aspects of the treatment of headache. Neurology, 1963, 13, 34-37.

Kudrow, L. The relationship of headache frequency to hormone use in migraine. Headache, 1975, 15, 36-40.

Lake, A., Rainey, J. & Papsdorf, J. Biofeedback and rational-emotive therapy in the management of migraine headache. Journal of Applied Behavior Analysis, 1979, 12, 127-140.

Lucas, R.N. Migraine in twins. Journal of Psychosomatic Research, 1977, 20, 147-156.

Luktev, E.R. Treatment of migraine headache by conditioned relaxation: A case study. Behaviour Therapy, 1971, 2, 592-593.

Meichenbaum, D. Cognitive factors in biofeedback therapy. Biofeedback and Self-Regulation, 1976, 1(2).

Mitchell, K.R. & Mitchell, D.M. Migraine: An exploratory treatment application of programmed behavior therapy techniques. Journal of Psychosomatic Research, 1971, 15, 137-157.

Paulley, J.W. & Haskell, D.A.L. The treatment of migraine without drugs. Journal of Psychosomatic Research, 1975, 19, 367-374.

Pearce, J. Migraine: A psychosomatic disorder. Headache, 1977, 17, 125-128.

Philips, C. Headache and personality. Journal of Psychosomatic Research, 1976, 20, 535-542.

Pichel, C.H. Treating migraine with TA and Gestalt: A case history. Transactional Analysis Journal, 1977, 7, 58-60.

Pardine, P. & Napoli, A. Personality correlates of successful biofeedback training. Perceptual and Motor Skills, 1977, 45(3), 1099-1103.

Rees, W.L. A controlled epidemiological study of the role of psychological factors in migraine. Archivos de Neurobiologia, 1974, 37, 243-251.

Reinking, R.H. & Kohl, M.L. Effects of various forms of relaxation training on physiological and self-report measures of relaxation. Journal of Consulting and Clinical Psychology, 1973, 43(5), 595-600.

Rose, F.C. Migraine - the Facts. New York: Oxford University Press, 1979.

Rotter, J.B. Generalized expectancies for internal versus external control of reinforcement. Psychological Monographs, 1960, 80(1 Whole No. 609).

Sachs, D.A., Talley, E. & Boley, K. A comparison of feedback and reinforcement as modifiers of a functional motor response in a hemiparetic patient. Journal of Behavior Therapy and Experimental Psychiatry, 1976, 1, 171-174.

Sachs, O. Migraine, Evolution of a Common Disorder. London: Faver and Faber, 1971.

Sandler, M. Transitory platelet monoamine oxidase deficit in migraine: Some reflections. Headache, 1977, 17, 153-158.

Saper, J.R. Migraine. American Medical Association Journal, 1978, 239, 2480-2484.

Sargent, J.D., Green, E.E. & Walters, E.D. Preliminary report on the use of autogemic feedback training in the treatment of migraine and tension headaches. Psychosomatic Medicine, 1973, 35(2), 129-135.

Sargent, J.D., Green, E.E. & Walters, E.D. The use of autogenic feedback training in a pilot study of migraine and tension headache. Headache, 1972, 12, 120-125.

Sehandler, S.L. & Grings, W.W. An examination of methods for producing relaxation during short-term laboratory sessions. Behavior Research and Therapy, 1976, 14, 419-426.

Schnarch, D.M. & Hunter, J.E. Personality differences between randomly selected migrainous and non-migrainous people. Psychotherapy: Theory, Research and Practice, 1979, 16(3), 297-309.

Schultz, J.H. & Luthe, W. Autogenic Therapy: Vol. I. Autogenic Methods, W. Luthe (Ed.), New York: Grune & Stratton, 1969.

Selby, G. & Lance, J.W. Observations on 500 cases of migraine and allied vascular headache. Journal of Neurology, Neurosurgery and Psychiatry, 1960, 23, 23-32.

Selye, H. The Stress of Life. New York: McGraw-Hill, 1976.

Selye, H. Stress Without Distress. Scarborough, New American Library; Signet Book, 1874.

Sicuteri, F., Testi, A. & Anselmi, B. Biochemical investigations in headache: increase in hydroxyindole acetic acid excretion during migraine attacks. International Archives of Allergy, 1961, 19, 55-58.

Silver, B.V., Blanchard, E.B., Williamson, D.A., Theobald, D.E., & Brown, D.A. Temperature biofeedback and relaxation training in the treatment of migraine headaches -- one-year follow-up. Biofeedback

and Self-Regulation, 1979, 4(4), 359-366.

Somerville, B.W. The influence of hormones upon migraine in women. Medical Journal of Australia, Special Supplement, 1972, 2, 6-11.

Sperling, M. A psychoanalytic study of migraine and psychogenic headache. Psychoanalytic Review, 1952, 39, 152-163.

Stoyva, J. Guidelines in the training of general relaxation. In J. Basmajian (Ed.), Biofeedback - Principles and practice for clinicians. Baltimore: Waverly, 1979.

Sturgis, E.T., Tollison, C.D., & Adams, H.E. Modification of combined migraine-muscle contraction headaches using BVP and EMG feedback. Journal of Applied Behavior Analysis, 1978, 11(2), 215-223.

Sulman, F.G. Migraine and headache due to weather and allied causes and its specific treatment. Upsala Journal of Medical Sciences Supplement, 1980, 31, 41-44.

Surwit, R.S. & Keefe, F.J. Frontalis EMG feedback training: An electronic panacea? Behavior Therapy, 1978, 9, 779-792.

Surwit, R.S., Shapiro, D. & Feld, J. Digital temperature autoregulation and associated cardiovascular changes. Psychophysiology, 1976, 13(1), 242-248.

Taub, E., & Emurian, C.S. Feedback-aided self-regulation of skin temperature with a single feedback locus. Biofeedback and Self-Regulation, 1976, 1(2), 147-168.

Touraine, G.A. & Draper, G. The migrainous patient. The Journal of Nervous and Mental Disease, 1934, 80, 1-23 and 183-204.

Trowbridge, L.S., Cushman, D., Gray, M.G. & Moore, M. Notes on the personality of patients with migraine. The Journal of Nervous and Mental Disease, 1943, 97, 509-517.

Turin, A. & Johnson, W.G. Biofeedback therapy for migraine headaches. Archives of General Psychiatry, 1976, 33, 517-519.

Waters, W.E. Migraine: Intelligence, social class and familial prevalence. British Medical Journal, 1971, 2, 77-81.

Waters, W.E. & O'Connor, P.J. Prevalence of migraine. Journal of Neurology, Neurosurgery and Psychiatry, 1975, 38, 613-616.

Weber, H. The psychological factor in migraine. British Journal of Medical Psychology, 1932, 12, 151-173.

Whatmore, G.B. & Kohli, D.R. The physiopathology and treatment of functional disorders. New York: Grune & Stratton, 1974.

Wickramasekera, I. A conditioned response model of the placebo effect: Predictions from the model. Biofeedback and Self-Regulation, 1980,

5(1), 5-15.

Wolff, H.G. Headache and Other Head Pain. New York: Oxford University Press, 1963.

Wolff, H.G. Personality features and reactions of subjects with migraine. Archives of Neurology and Psychiatry, 1937, 37, 895-921.

Yates, A.J. Biofeedback and the modification of behavior. New York: Plenum Press, 1980.

Ziegler, D.K. The epidemiology and genetics of migraine. Research and Clinical Studies in Headache: 5, 21-33 (Karger, Basel, 1978).

REFERENCE NOTE

1. University of Alberta, 1980-81, developed by the researchers involved in the migraine headache study under the direction of Dr. G.W. Fitzsimmons.

APPENDIX A

Inclusion Criteria

Name: _____ Address: _____

Phone: _____ Sex: _____

1. What is your age? ____(18 to 59)
2. How often have your headaches occurred in the last 2 months? ____(less than 3X/day & greater than 1/mo.)
3. Do you take any medication for your headache? _____

   What is the name of the medication? _____

   How well does it control headache? _____(Doesn't)

   Currently using oral contraceptives? ____; pregnant? ___(no)

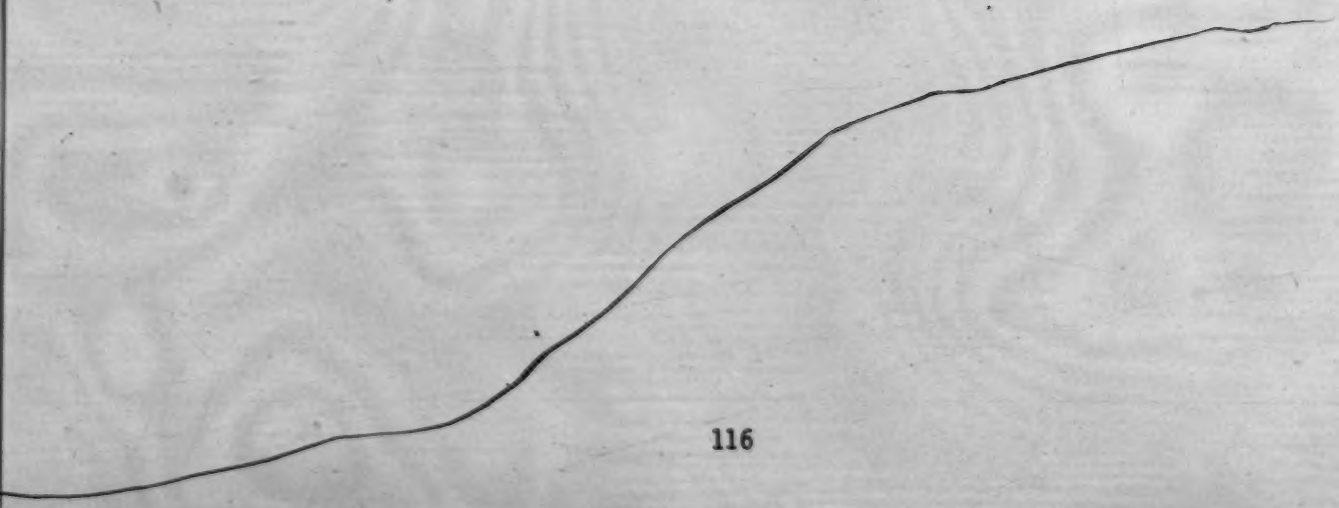
   Are you on any other medications? Specify: _____
4. Have your headaches occurred one or more times per month over the past 2 years? _____(yes)
5. Are you currently receiving any form of psychotherapy? _____(no)
6. Do you ever experience sensory losses or paralysis of some muscles during a headache? _____(no)
7. Do you suffer from a convulsive disorder (epileptic seizures)? _____(no)
8. Do you have any form of heart disease or disorder? _____(no)
9. Do you have any health problems such as diabetes, hypertension, etc? Specify: _____

SUBJECT MUST REPORT "YES" TO THREE OF THE FOLLOWING ITEMS:

1. Does the head pain sometimes exist on one side of the head only?
2. Is the head pain generally pulsative (or throbbing)?
3. Does nausea or vomiting generally accompany the headache?
4. Does sensitivity to light generally accompany the headache?
5. Has your headache been diagnosed as a migraine by your physician?

(see Reference Note 1)

APPENDIX B



Treatment Contract

Participant - I understand that my participation in the migraine treatment program will require my full cooperation in each of the following components of the study:

1. Punctual attendance at all treatment sessions scheduled;
2. Attendance at one pre-training orientation session held several days before treatment and one post-treatment follow-up session held one month after the treatment period;
3. One half-hour daily practice of specific relaxation skills learned in treatment to continue throughout the training period;
4. Hourly monitoring of headache activity and medication consumption prior to and throughout the training period;
5. Keeping a headache diary for one year following the treatment period;
6. Notifying your counsellor by phoning 432-5214 (during regular office hours) if unable to keep a scheduled appointment.
7. Notifying the Department of Educational Psychology, 6th floor-Education North, University of Alberta should your address change.

Date: _____ Signature: _____

Counsellor - I promise that all records of Participants: names, addresses and personal information will be kept confidential. At the completion of this study a summary of the results obtained shall be made available to all those who fully participated.

Date: _____ Signature: _____

(see Reference Note 1)

APPENDIX C

Medical Form

Name of Physician: _____

Name of Patient: _____

Date of Birth: _____

Address: _____

_____

Phone: _____

The above named patient has been selected to participate in a treatment program for migraine patients being conducted at the University of Alberta, Department of Educational Psychology. This research is being supervised by Dr. George Fitzsimmons. The techniques being used may include relaxation training and psychophysiological monitoring including electromyography, galvanic skin response, and surface skin temperature.

We are requesting each patient to obtain the signature of their physician to verify that they have received a recent medical examination and to ensure that there is no medical reason why they should not participate in the research project.

For Physician

(A) This is to certify that _____ has been medically examined and I do not advise against his/her participation in the program described.

(B) I _____ (do, do not) agree that the headache pain which this person reports is of the migraine form.

Date: _____

Physician's Signature: _____

(see Reference Note 1)

APPENDIX D

Psychophysiological Profile Procedure

1. Record room temperature.

2. Seat subject in recliner chair and tilt chair to the first recliner position. Inquire whether a headache is now present and if so reschedule the session.

3. Hook-Up GSR - dominant hand; Temperature - nondominant hand; EMG - frontal area with electrodes spaced at 1 inch.

4. Explanation "Today's session will last approximately 40 minutes. What I am going to do is attach you to 3 biofeedback instruments in order to see what levels of activity you produce in 3 different physiological systems: (1) skin temperature, (2) muscle tension, and (3) skin perspiration (indicate corresponding instrumints). These instruments will not shock you or harm you in any way, they merely attach onto the surface of your skin with these wires. We are hooking you up today in order to find out how your body activity corresponds to your headache pattern, and how the relaxation treatment program changes both your body activity and your headache pattern. Do you have any questions?"

5. Relaxation instructions to be read to the subject. "For the next 5 minutes I would like you to relax comfortably with your eyes open and just listen to the music being played in the background. Try to avoid unpleasant thoughts and just enjoy this 5 minutes of rest. After 5 minutes have elapsed I will ask you to sit for 15 minutes with your eyes closed. Please sit quietly without moving or talking and keep your hands on the arm rest with your palms facing upward. Do you have any questions? Okay then, starting with your eyes open just relax and I will tell you when 5 minutes are up.

6. Turn On EMG, GSR, Temperature, 3 Optical Isolators, Computer, and Printer.

7. Settings EMG scale X1; GSR scale X1; Temperature - center needle.

8. Identify subject on ticker tape.

Example:		
	Name:	John Headache
	Date:	May 30, 1981
	Therapist's Initials:	P.F.
	Initial Temperature Setting:	72

During session draw lines to divide "A" - 5 minutes adaptation; "B" - 15 minutes relaxation; "C" - 3 minutes stress; "D" - 5 minutes recovery. Label periods between lines on the ticker tape.

9. Turn on Music low volume.

10. Start Timer and Computer Conduct 5 minute adaptation.

Reset temperature where necessary but only while printer is operating. Write in new value on the printout. Use a dashed line to mark where new value begins on printout.

After 5 minutes draw a line on the printout and mark "B" in next section.

Reset timer for 15 minutes.

11. Relaxation period Say to subject: "And now I would like you to sit for 15 minutes with your eyes closed. Try not to fall asleep."

    After 15 minutes draw a line on the printout and mark "C" in the next section.

    Reset timer for 3 minutes.

12. Stress period Say to the subject: "Okay while keeping your eyes closed now I want you to perform a mental task for me. I want you to subtract 7 from 1000 and then to continue subtracting 7 from your answer as fast as possible until I tell you to stop. Then I will ask you what number you got to. Do this in your head not out loud. Okay so 1000 minus 7 is ... now keep going, to yourself."

    After 3 minutes say "Stop".

    Draw a line on the printout and mark "D" in the next section.

    Ask: "What number did you get to?"

    Reset timer for 5 minutes.

13. Recovery Period Say to client: "Now I just want you to relax again with your eyes closed and listen to the music without interruption for 5 minutes and then we are finished.

    After 5 minutes end session and disconnect subject.

    Graph data and check batteries.

APPENDIX E

ONLY COPY AVAILABLE
SEULE COPIE DISPONIBLE

Headache Rating Scale

NAME: _____

To monitor your headache level mark the appropriate number on the graph at each hour and join the points together. Placing the coloured dot on your watch will help you remember to do this.

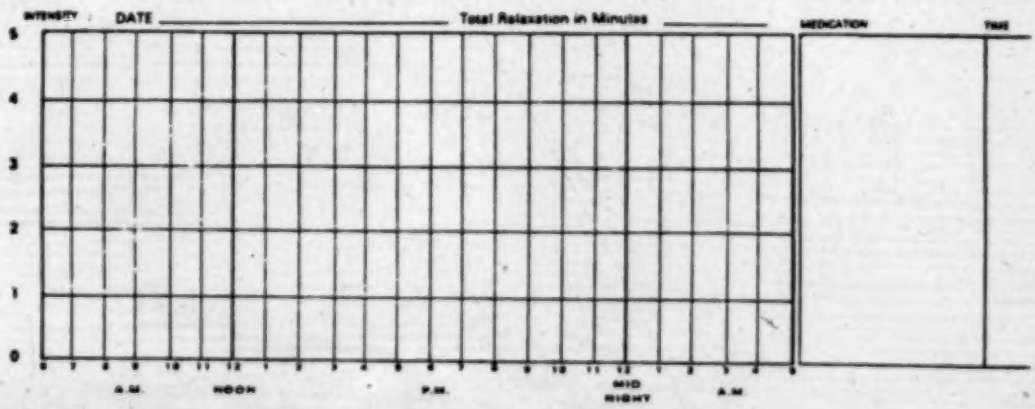
0 — No headache.
1. — Low level, only enters awareness when you think about it.
2 — Aware of headache most of the time but it can be ignored at times.
3 — Painful headaches but still able to continue job.
4 — Severe headache, difficult to concentrate with understanding tasks.
5 — Intense incapacitating headache.

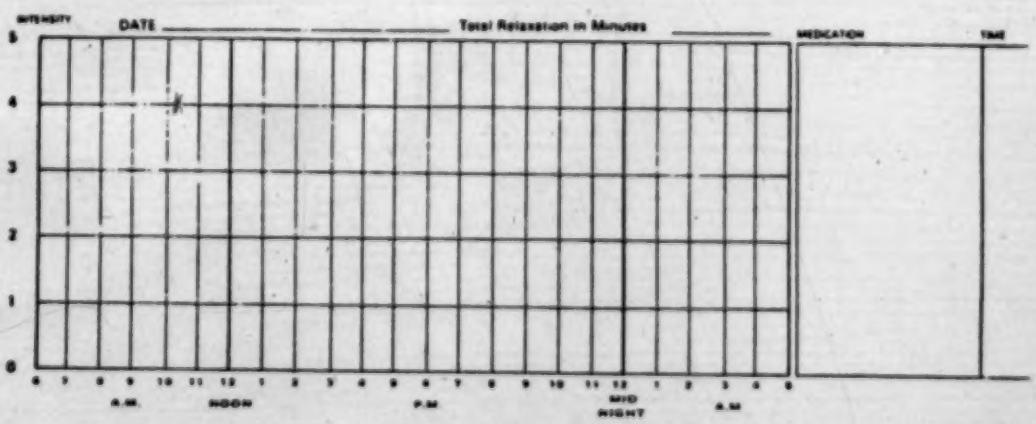
It is important to monitor the intensity of your headaches for at least two reasons:

1. Research has shown that this will help to reduce the psychological side effects that often accompany a headache.
2. It is useful in helping to determine the effects of your treatment program.

The following five point scale is useful in helping people monitor the severity of their headache.

PROGRESS CHART





APPENDIX F

EMG Training Rationale

The treatment sessions you are receiving are designed to teach you how to produce more effective physiological relaxation at will. Your final goal in treatment is to become able to discriminate excessive stress in your body and be able to remove such stress in order to prevent migraine headaches. Regular and consistent practice at removing excessive stress will eventually develop into a life-style habit. When this occurs your body will maintain a more relaxed level of arousal without conscious effort. It may take somewhere between a couple of weeks to several months to develop this automatic habit, depending upon the amount of relaxation practice you do and the strength of the stress habit you now have.

In biofeedback training you will learn to relax efficiently, guided by the feedback signal. The idea is to decrease your muscle tension voluntarily as you relax and learn to use decreased muscle tension as an index of your relaxation level. Over time you will learn to produce greater levels of relaxation in less time and to maintain these levels for longer periods. Even though the biofeedback is only attached to the head region it is to your advantage to learn to decrease your muscle tension as part of a total body relaxation response.

Biofeedback guided relaxation takes place in three stages. The first stage is called the "awareness" stage where your brain is merely made aware of how much feedback corresponds to how much muscle tension. Gradually the second stage emerges where in addition to becoming aware of tension levels you become able to control the tension and further reduce it. This second stage is known as the "control" stage.

Please note that the control stage takes time to emerge because you must learn the skill involved. Also note that contrary to most other intentional learning you do, learning to relax does not involve active striving. The more you strive the more tense you will become. Instead of actively striving to reduce muscle tension you must passively concentrate on the feedback signal and "allow" the tension to reduce. In other words, "let it happen".

The final stage of biofeedback guided relaxation, following awareness and control is the "weaning" stage. Weaning involves practice at producing the relaxation response in the absence of the biofeedback signal. Such practice will begin once you have learned the relaxation response. In this way you can learn an effective relaxation skill which is not dependent upon biofeedback.

Many persons have asked what thinking strategies they should be using to decrease muscle tension as they passively concentrate. Other than advising such persons to avoid unpleasant thoughts or stress-related rumination there is no particular strategy that everyone will find effective. Some people use mental images of relaxing settings such as laying on a warm beach, skiing down a mountain in slow motion, or watching a beautiful sunset. Others think suggestive phrases to them-

selves such as "I am becoming warm and relaxed". Others do not think about anything, they let their minds go blank. Most people find some particular strategy useful at first but as they learn to relax efficiently, letting go of tension becomes a skill they can utilize without any conscious strategy. Over the course of the training sessions, you should use whatever strategies you feel comfortable with to relax. But remember, the important thing is not to force any approach or to try too hard, because effort is the opposite of relaxation. Just let the approach you choose flow, just imagine it is already happening.

(see Reference Note 1)

APPENDIX G

Digit Temperature Training Rationale

The treatment sessions you are receiving are designed to teach you how to produce more effective physiological relaxation at will. Your final goal in treatment is to become able to descriminate excessive stress in your body and be able to remove such stress in order to prevent migraine headaches. Regular and consistent practice at removing excessive stress will eventually develop into a lifestyle habit. When this occurs your body will maintain a more relaxed level of arousal without conscious effort. It may take somewhere between a couple of weeks to several months to develop this automatic habit, depending upon the amount of relaxation practice you do and the strength of the stress habit you now have.

In biofeedback training you will learn to relax efficiently, guided by the feedback signal. The idea is to warm your hands voluntarily as you relax and learn how to use hand warming as an index of your relaxation level. Over time you will learn how to produce greater levels of relaxation in less time and to maintain these levels for longer periods. Even though the biofeedback is only attached to one of your fingers it is to your advantage to learn how to warm as part of a total body relaxation response.

Biofeedback guided relaxation takes place in three stages. The first stage is called the "awareness" stage where your brain is merely made aware of how temperature changes correspond to vascular changes brought about by stress and relaxation. Gradually the second stage emerges where in addition to becoming aware of stress levels you become able to control the stress and further reduce it. This second stage is known as the "control" stage.

Please note that the control stage takes time to emerge because you must learn the skill involved. Also note that contrary to most other intentional learning you do, learning to relax does not involve active striving. The more you strive the more tense you will become. Instead of actively striving to warm your hands you must passively concentrate on the feedback signal and "allow" the warming to occur. In other words, "Let it happen".

The final stage of biofeedback guided relaxation awareness and control is the "weaning" stage. Weaning involves practice at producing the relaxation response in the absence of the biofeedback signal. Such practice will begin once you have learned the relaxation response. In this way you can learn an effective relaxation skill which is not dependent upon biofeedback.

Many persons have asked what thinking strategies they should be using to induce hand warming as they passively concentrate. Other than advising such persons to avoid unpleasant thoughts or stress-related ruminations there is no particular strategy that everyone will find effective. Some people use mental images of relaxing settings such as lying on a warm beach, skiing down a mountain in slow

motion, or watching a beautiful sunset. Others think suggestive phrases to themselves such as "I am becoming warm and relaxed". Others do not think about anything, they let their minds go blank. Most people find some particular strategy useful at first but as they learn to relax efficiently, letting go of tension becomes a skill they can utilize without any conscious strategy. Over the course of the training sessions, you should use whatever strategies you feel comfortable with to relax. But remember, the important thing is not to force any approach or to try too hard, because effort is the opposite of relaxation. Just let the approach you choose flow, just imagine it is already happening.

(see Reference Note 1)

APPENDIX H

E.M.G. Training Procedures and Instructions

1. Check tape in printer.

2. Take room temperature.

3. Collect forms from client.

4. Mark treatment session number in progress in file and on printout.

5. (a) Attach EMG electrodes. Make sure impedence levels read less than 1 for each electrode with scale setting at X30. If not re-do cleansing of forehead with alcohol.

   (b) Attach temperature thermister to the palmer surface of the second phalange of the middle finger on the nondominant hand.

6. Check if client has read the rationale, if not, have them read it.

7. Settings: Center temp. needle and record setting on printout. Leave EMG at X1 setting. Explain EMG gauge setting and units of measurement to clients. Occlude all gauges from client's view and disengage auditory feedback.

8. Turn on: EMG, Temp., Optical Isolators, Computer and Printer. Turn fluorescent lights off.

9. Identify Client on ticker tape.

   Example:

Room temp:	72°
Name:	John Headache
Date:	May 30, 1980
Therapist's Initials:	P.J.C.
Initial Temp Setting:	82°
Session:	Rx #1

10. Give the following instructions: This session will last approximately 40 minutes. Please keep your eyes open during the entire session. The session will consist of 4 phases. You will not receive any biofeedback during the first three phases. The first phase is an adaptation phase. For the next 5 minutes please just sit in the chair with your eyes open.

11. Start timer and computer simultaneously.

12. After 5 minutes draw a line on the printout and say "For the next 2 minutes I will be collecting baseline data. Please continue to sit quietly without talking and keep your eyes open."

13. After 2 minutes draw a line on the printout and say "For the next 2 minutes I would like you to decrease your muscle tension."

14. After 2 minutes draw a line on the printout and say: "This is the training phase. You will have three periods of 5 minutes of practice and 1 minute of rest. Uncover the EMG gauge. Have client put on headphones and turn up the volume to the preferred setting. Say: As you decrease the muscle tension in your head region the clicks will slow down. For the next 5 minutes I would like you to practice decreasing your muscle tension."

15. Draw a line on the printout and begin timer.

16. After 5 minutes draw a line on the printout and say "Please stop practising now and just take a break." Discuss performance. After 1 minute draw a line on the printout and say: "Now please begin practising again for 5 minutes." Follow with one minute of rest.

17. Follow with 5 more minutes of practice.

Note: The order of items 18 and 19 can be conducted interchangeably so when subjects are seen in pairs, one person may be given 18 while the other does 19.

18. Conduct Written Summaries Procedure. At the end of each session discontinue biofeedback monitoring and give each subject the following instructions: "Please spend 5 minutes writing down a description of the strategies which you employed to relax and also identify any feelings or sensations which appeared to be associated with slower clicking. A new summary will be written each session and taken home with you until the next session at which time we would like you to hand it in for our records. Go ahead now, I will tell you when 5 minutes have elapsed."

After the 5 minute period give the following instructions: "For training to be effective in suppressing migraines you must practise relaxation daily utilizing the strategies you have written down. In this manner you will be attempting to duplicate outside the lab. the same feeling state associated with slow clicking biofeedback.

You will be asked to use the strategy to aid your relaxation during the final part of the relaxation tape. You are also asked to use the strategy to help you relax whenever you experience the prodrome (aura) or feel that you will soon get a migraine and also anytime during the day that you feel you are stressed or overly tense. Please remember to monitor the total minutes of daily relaxation you practise on your headache monitoring forms.

19. Discuss Progress During Session. At the end of each training session show each subject the EMG levels they achieved and compare these to the two-minute EMG average value computed at the end of 15 minutes of relaxation during the pretreatment monitoring session. Point out that ideally they will be learning to become more relaxed, faster, and be able to maintain such relaxed levels longer.

20. Discuss Medication. After each treatment session discuss medication consumption with the subject. Subjects should be advised and repeatedly reminded to monitor their medication intake and to consult with their physicians about any changes required in their prescriptions. Inform subjects that increased relaxation may alter the effects of their medication, migraine or otherwise. This is especially true for subjects taking medication for hypertension, or diabetes.

(see Reference Note 1)

APPENDIX I

Biotic Band Monitoring and Recording

Please use your Biotic Band device to monitor finger temperature daily while you listen to the relaxation tape. Attach the band to the middle finger of your non-dominant hand. Place the band with the temperature scale on the palmer surface of your finger and center it mid-way along the length of your finger. The band should be snug but not tight. While relaxing try to sit in a comfortable chair with arm rests so that your hand temperature will not be effected by warmth from your lap.

As you practise relaxation note how your finger temperature increases. On your headache monitoring form, write down your finger temperature: (a) after the band has been on your finger for 1 minute and before you start the tape, and (b) at the end of the tape.

Please avoid crushing or crumpling the band as they may become inaccurate with abuse. If you think that your band has broken bring it in to your next training session. The bands must be returned at the end of treatment.

BIOTIC-BAND II has a range of 20.0 F divided into two degree intervals which are indicated on the band by the printed numbers. The liquid crystal squares beside the numbers light up when the temperature of the finger being monitored comes within that two degree range. Within each range of two degrees, color changes indicate smaller changes in the temperature. Each color change equals a change of 0.5 F as shown in the table below.

Lighted Degree	Red-Tan	Orange	Yellow-Green	Blue-Green	Blue
78	78	78.5	79	79.5	80
80	80	80.5	81	81.5	82
82	82	82.5	83	83.5	84
84	84	84.5	85	85.5	86
86	86	86.5	87	87.5	88
88	88	88.5	89	89.5	90
90	90	90.5	91	91.5	92
92	92	92.5	93	93.5	94
94	94	94.5	95	95.5	96
96	96	96.5	97	97.5	98

In taking a reading always read the highest temperature showing. The purple color which may sometimes be visible on some squares should always be ignored.

APPENDIX J

Temperature Training Procedure and Instruction

1. Check tape in printer.

2. Take room temperature.

3. Collect forms from client.

4. Mark the treatment session number in progress in the file and on the printout.

5. (a) Attach EMG electrodes. Make sure impedence levels read less than 1 for each electrode with scale setting at X30. If not re-do cleansing of forehead with alcohol.

   (b) Attach temperature thermister to the palmer surface of the second phalange of the middle finger on the non-dominant hand.

6. Check if client has read the rationale, if not, have them read it.

7. Ask client what they think their hand temperature is.

8. Settings: Center temp. needle and record setting on printout. Leave EMG at X1 setting. Explain temp. gauge setting and units of measurement to clients. Tell client what their hand temp. is. Occlude all gauges from client's view and disengage auditory feedback.

9. Turn on: EMG, Temp., Optical Isolators, Computer, and Printer. Turn fluorescent lights off.

10. Identify client on ticker tape.

    Example:

Room temp:	72°
Name:	John Headache
Date:	May 30, 1980
Therapist's Initials:	P.J.C.
Initial Temp. Setting:	82°
Session:	R_x#1

11. Give the following instructions: This session will last approximately 40 minutes. Please keep your eyes open during the entire session. The session will consist of 4 phases. You will not receive any feedback during the first 3 phases. The first phase is an adaptation phase. For the next 5 minutes please just sit in the chair with your eyes open.

12. Start timer and computer simultaneously.

13. After 5 minutes draw a line on the printout and say "For the next 2 minutes I will be collecting baseline data. Please continue to sit quietly without talking and keep your eyes open.

14. After 2 minutes draw a line on the printout and say "For the next 2 minutes I would like you to increase your temperature."

15. After 2 minutes draw a line and say: "This is the training phase. You will have three 5 minute trials of alternately practising warming and cooling your temperature 2°. Uncover the temperature gauge. Have the client put on the headphones and turn up the volume to the preferred setting. Say: "As you increase your temperature the tone will change. If you are at or above 90° please begin by cooling 2°. If you are below 90° please begin by warming 2°. Once you have warmed or cooled 2° please maintain that temperature for the remainder of the 5 minute period. Draw an arrow on the printout indicating whether the client is warming (→) or cooling (←). Use a block on top of the machine to show the client the direction the needle should go.

16. Draw a line on the printout and begin timer.

17. After 5 minutes draw a line on the printout and ask client to continue to warm if they have not reached 90° or reverse directions.

18. Continue the same procedure for one more 5 minute period.

    Note: The order of items 19 and 20 can be conducted interchangeably so when subjects are seen in pairs one person may be given 19 while the other does 20.

19. Conduct Written Summary Procedure. At the end of each biofeedback session, discontinue biofeedback monitoring and give subjects the following instructions: "Please spend 5 minutes writing down a description of the strategies which you employed to relax and also identify any feelings or sensations which appeared to be associated with hand warming. A new summary will be written each session and taken home with you until the next session at which time we would like you to hand it in for our records. Go ahead now, I will tell you when 5 minutes have elapsed."

    After the 5 minute period give the following instructions: "For training to be effective in suppressing migraines you must practise relaxation daily utilizing the strategies you have written down. In this manner you will be attempting to duplicate outside the lab the same feeling state associated with hand warming biofeedback in the lab.

You will be asked to use the strategy to aid your relaxation during the final part of the relaxation tape. You are also asked to use the strategy to help you relax whenever you experience the prodrome (aura) or feel that you will soon get a migraine and also anytime during the day that you feel you are stressed or overly tense. Please remember to monitor the total minutes of daily relaxation you practise on your headache monitoring forms.

20. Discuss Progress During Session. At the end of each training session show the subject the minute by minute temperature levels they achieved. If these levels at any time were higher than the temperature recorded after 15 minutes of relaxation during the profile session, then indicate this to the subject. Otherwise do not. Point out that ideally they will be learning to become more relaxed, faster, and be able to maintain such relaxed levels longer.

21. Discuss Medication. After each treatment session discuss medication consumption with the subject. Subjects should be advised and repeatedly reminded to monitor their medication intake and to consult with their physicians about any changes required in their prescriptions. Inform subjects that increased relaxation may alter the effects of their medication, migraine or otherwise. This is especially true for subjects taking medication for hypertension or diabetes.

(see Reference Note 1)